Clinical Lectures

ON

Diseases of the Heart, Lungs and Pleura

—PATTON

J. C. Hughes M. D,
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J. F. Hughes

CLINICAL LECTURES

ON

DISEASES OF THE

HEART, LUNGS AND PLEURA

DESIGNED FOR THE USE OF PRACTITIONERS AND
ADVANCED STUDENTS OF MEDICINE.

BY

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SECOND EDITION—REVISED AND ENLARGED.

PREFACE TO THE SECOND EDITION.

The rapid exhaustion of the first edition of these lectures has left little time for new developments in the field of medicine which they cover. However, such matters of interest in this connection as have developed in the interval of the publication of the first edition have been incorporated in this issue. The classification and general arrangement of matter remains as in the first edition, but the text has been thoroughly revised in conformity with the original intention of conciseness and practical clinical utility.

Chapter I has been augmented by the addition of matter which completes the subject of diseases of the pericardium. The rhythmic disturbances of the heart have received fuller consideration, principally through a more complete delineation of tachycardia and the clinical features of heart hurry. Changes have been made in the mechanical arrangement of subject-matter which, it is believed, will enhance the convenience of perusal, as well as improve the appearance of the text.

Some illustrations, mostly original, have been added in the hope that they will emphasize diagnostic features on which stress is placed in the text.

J. M. P.

October, 1900.

PREFACE TO THE FIRST EDITION.

This series of lectures, rearranged and somewhat ampli-fied, have been republished with the hope that in a more perma-nent form they may be of greater value to both practitioner and student.

The subjects contained herein have been considered solely from a clinical standpoint, with the advantage of brevity con-stantly in mind, and with the object of presenting pertinent matter without entailing much expenditure of the reader's time. In arriving at this object what little of literary merit they may have contained has frequently been sacrificed to conciseness, and if the latter has not been carried to the point of ambiguity such deference to succinctness may be excused.

While fully appreciating the many demerits of these lec-tures they are yet issued in the faith that there may be com-pensation for their faults in what little of value they may contain.

J. M. P.

September, 1899.

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CHAPTER I.

PERICARDITIS.

Pericarditis is of more frequent occurrence than is generally supposed. It is very liable to pass unrecognized because of the greater prominence of associated conditions.

As a secondary affection it may come on insidiously, and this fact together with the difficulties attendant upon the diagnosis of pericardial inflammation causes it, in many instances, to be overlooked during life.

Pericarditis may occur at any age. Behier cites a case occurring in a child eleven months old. Pericarditis may be acute, or chronic as a sequel to the acute attack. It is probably never idiopathic, though some authorities claim that it may be. The relative frequency of the several varieties may be indicated by the following series of 324 cases from the Charité Hospital, Berlin: Sero-fibrinous, 108; hæmorrhagic, 30; purulent, 24; tubercular (secondary), 24; tubercular (primary), 2; partially adherent, 111; totally adherent, 23; ossified, 2.

ÆTIOLOGY.—Pericarditis is most frequently associated with rheumatism, pneumonia or nephritis. According to Bamberger's statistics thirty per cent. of the cases are rheumatic. In rheumatism pericarditis is of early occurrence and may develop before or during the first week of the joint symptoms. The number and severity of the rheumatic manifestations do not appear to have any constant relation to the liability of pericardial involvement, or to the nature of the inflammatory process, though the exudation is generally plastic or fibrinous.

Occurring with pneumonia pericarditis is usually a latent affair. Shattuck found thirteen cases of pericarditis in twenty deaths from pneumonia. In eight per cent. of these cases there were no signs of pericarditis previous to death. In one hundred autopsies on pneumonic cases Osler found five cases of pericarditis.

In pericarditis secondary to pneumonia the exudation is

likely to be purulent. Of late years the tendency for purulent inflammations of both pleura and pericardium to complicate the influenza pneumonias has been quite marked.

In Bright's disease pericarditis is usually of late occurrence, and is often a forerunner of the general uræmic state.

Statistics give pericarditis as associated with Bright's disease in from 4 to 14 per cent. of the cases of nephritis. It is equally common in connection with parenchymatous nephritis and renal cirrhosis (Grainger Stewart). Again, it is said to be more common in connection with interstitial nephritis (Lecorché and Talamon). Pericarditis does not occur with chronic rheumatism and its association with gout is doubtful.

Acute alcoholism may cause pericarditis or it may be secondary to the zymotic diseases, or may be associated with scurvy, enteric fever or pyæmia.

Pericarditis may be secondary to disease of contiguous tissues, as in tuberculosis, inflammations or morbid growths in the lungs, pleuræ, or mediastinum, or to inflammation of the abdominal surface of the diaphragm, perihepatitis or abscess of the liver.

Traumatism through the chest wall or through the œsophagus may cause pericarditis. (Bruist and Flint report cases of fatal pericarditis from swallowing false teeth plates.)

A fact that I would particularly emphasize is the frequent association of pericarditis with other cardiac lesions, particularly with endocarditis in children.

Sturges, in commenting on the frequency of pericarditis in children, either alone or in combination with endocarditis, states that the latter without the former is rare in childhood. In one hundred cases of fatal heart disease Sturges found only six showing no evidence of pericarditis.

Broadbent states that in eighty-six fatal cases of heart disease, thirty-four showed evidence of pericarditis, and in thirty-one it was associated with valvular disease. Osler thinks that tuberculosis ranks close to rheumatism as a cause of pericarditis. Males appear to suffer oftener than females and there is no age limit.

MORBID ANATOMY.—If you recall the anatomy of the pericardium you will remember that it is a closed sac enveloping

the heart, composed of two layers, an external fibrous and an internal serous layer. The fibrous layer is attached below to the central tendon of the diaphragm. Above, both layers envelop the great vessels extending well up towards the beginning of the transverse portion of the aortic arch. The visceral layer of the serous coat is that which is reflected over the heart itself.

Inflammation of the pericardium may affect a portion or the whole of the pericardial surfaces. It may be, and frequently is, limited to that portion surrounding the origin of the great vessels, or it may affect in spots any portion of the pericardium. When the inflammation is fibrinous in character and limited in extent it is apt to occur at the base of the heart about the root of the great vessels or along the transverse groove.

In Bright's disease we often find the most marked changes about the under surface of the right ventricle. Here we may find smooth, white, milky, opaque patches caused by thickening of the serous coat. These spots are called *maculae albidae*. They have caused much discussion, but have probably only a pathological interest. Roberts thinks they are caused by mechanical irritation from the movements of the heart; and that they may in some instances cause a limited friction sound. There may be cartilaginous plates.

The nature of the inflammatory exudate will be serous, fibrinous or purulent; any of these may be hæmorrhagic in character. The serous form occurs in persons whose vitality and resistance is naturally low, or has been reduced by some immediately preceding or concomitant illness. The local injury to the tissues is slight; or, in persons whose vitality is much reduced, an inflammation of considerable intensity may result in serous exudation.

In serous effusion the normal transudation from the vessels is increased with some excess of albumen. It contains very few leucocytes and does not coagulate, or, at most, only a few flakes may be found.

The quantity of the exudate will vary from a few drams to that sufficient to distend enormously the pericardial sac.

With slightly more intense injury we may have fibrin

poured out with the serous effusion and we may find flakes,
flat or round masses of fibrin, floating free in the serous effusion,—*sero-fibrinous effusion*. There is usually, however,
more or less fibrin suspended in all serous effusions.

Serous effusion may be green, yellow, brown or red in
color.

In fibrinous inflammation, as has been stated, there is
more intense injury to the tissues. The endothelial lining is
damaged. The exudation is more albuminous and is rich in
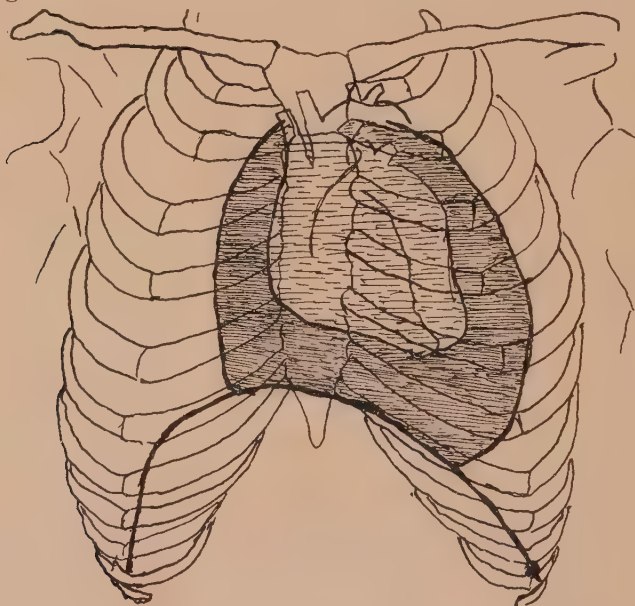
Diagram of pericardial sac, filled with sero-fibrinous effusion. Depression of diaphragm with partial obliteration of Traube's semilunar
space.

leucocytes. It is very coagulable and masses of lymph accumulate on the surface of the inflamed area. This lymph is composed of leucocytes entangled in the meshes of fibrin.

The leucocytes may be few and be surrounded by a granular or fibrillated matrix. (*Pericarditis sicca.*)

If the process be limited to a small area the opposite
surface may become involved by contact and local adhesions
may form.

These bands may constrict the great vessels and be the

cause of murmurs during life. The layer of fibrinous deposit is apt to be thickest on the parietal layer of the pericardium. If the process involves the whole pericardial surface adhesion of a portion of the pericardial layers may result, or complete obliteration of the pericardial sac may occur (*adherent pericardium*). As a result of imperfect absorption and the degeneration of inflammatory products with tissue organization, the heart may become surrounded with a mass of calcareous plates which from mechanical reasons and from changes induced in the heart itself may result in failure of that organ.

In fibrinous pericarditis when the exudation of lymph is plentiful, upon separating the pericardial surfaces they may present a rough, hairy appearance and to touch a sensation likened to that of the dorsal surface of a beef's tongue (*cor hirsutum, cor villosum or tomentosum*). This condition develops within two or three days after the beginning of the inflammation, as in the following case, seen in consultation.

Boy aged 9 years. Rheumatism for two weeks. Mitral regurgitation. On fifteenth day developed pain over heart with dry, rubbing, to-and-fro friction sound. Death on eighteenth day. Autopsy showed acute endocarditis. Incompetent mitral valve. Dilated heart cavities. Recent plastic pericarditis. The entire heart covered by the visceral layer of the pericardium had a sticky, hairy appearance and feel. It was covered with villosities and vegetations from one-eighth to one-fourth of an inch in height. No fluid in the pericardial sac.

The purulent form of pericarditis is secondary to the acute forms or to empyema, caries of the ribs, suppurative mediastinitis, gangrene of the lungs, or may be traumatic or metastatic. It is never primary. In purulent inflammation the injury is intense and prolonged. The exudation contains the same elements as in the fibinous form but no coagulation occurs; no lymph forms and new vessels are not developed.

Serous and fibrinous stages often precede the purulent stage of inflammation.

Putrefactive changes may occur if drainage is imperfect

or if air enters the pericardium through adhesions and perforation between it and the œsophagus, lungs, stomach or intestines.

Hæmorrhagic pericarditis as a type of inflammation is rare. Large numbers of red blood corpuscles may escape with the exudate or newly formed vessels may rupture, giving a hæmorrhagic character to any exudation. In purpura, scurvy, tuberculosis or malignant disease, however, there may be sufficient escape of blood to constitute a hæmorrhagic type of inflammation.

One of the most important features of pericardial inflammations is their effect on the heart itself.

Every pericardial inflammation of any severity is probably accompanied by more or less myocarditis involving the immediately adjacent muscular tissues which may be infiltrated by small cells. The fibres themselves may have undergone granular degeneration or coagulation necrosis. Fibroid induration of the heart may result from pericarditis, especially, according to Fagge, in association with syphilis accompanied by vascular lesions. When due to pericarditis the fibroid change is in the external portion of the walls of the heart and both ventricles are affected alike.

Enlargement of the heart is of frequent occurrence secondary to pericarditis, especially when there have been extensive adhesions of the pericardial surfaces. This enlargement has been attributed to the extra labor thrown on the heart by the adhesions interfering with its action and was supposed to occur independently of valvular lesion (Chevers, Barlow, Hope).

Some authorities claim that as a sequence of pericarditis cardiac enlargement does not occur independently of valvular lesion (Stokes, Sibson, Balfour).

All cases of adherent pericardium do not cause cardiac enlargement, nor are all cases of cardiac enlargement, which occur in connection with pericarditis, associated with valvular lesions. Broadbent's explanation of this fact is that the heart becomes dilated during an attack of pericarditis, from weakness of the heart muscle. Before it regains its usual condition pericardial adhesions develop which maintain it in an enlarged

state. Hypertrophy then develops in compensation to the dilatation. In those cases where enlargement does not take place, contraction of the heart occurs before the adhesions form.

Adherent pericardium may be associated with mediastinitis of varying degree. According to Harris there are three classes of cases: The first comprises adherent pericardium with fibrinous increase in the tissues of the mediastinum and adhesion of the external layer of the pericardium to surrounding tissues, with or without caseation of the mediastinal glands,—*indurative mediastino-pericarditis.*

The second class comprises adherent pericardium, adhesion of the external layer to surrounding parts, no general mediastinitis,—*pericarditis externa and interna.*

The third class comprises increase of fibrous tissue in the mediastinum, no adherent pericarditis internally,—*chronic mediastinitis.*

CLINICAL HISTORY.—The clinical history of pericarditis is indifferent as it is always mixed with the history of associated morbid conditions. There are three rational symptoms, however; pain in the præcordium, palpitation or cardiac arrhythmia and dyspnœa. The pain may or may not be severe, it may be confined to the cardiac region or may extend to the brachial plexus and down the left arm. Pressure on the liver against the diaphragm increases the pain. With the palpitation there is a sense of constriction and distress in the chest. Usually there is some cough. The dyspnœa depends largely on the amount of effusion. In cases of fibrinous exudation there is rapid and jerky respiration. If the effusion is large there may be orthopnœa.

The pulse will range from 100 to 130 in plastic pericarditis, and is strong and full. With effusion it becomes feeble or suppressed, but rapid.

In large effusions the pulse becomes irregular or intermittent and is markedly dicrotic; it is not in proportion to the action of the heart. The temperature usually rises to 100°–101°; in some cases it may go much higher.

In acute pericarditis the patient assumes a recumbent position, somewhat more toward the right than the left side. When there is much effusion the patient wishes to have the

shoulders elevated and cannot lie down. The face will have
an anxious, apprehensive expression. Syncope is apt to come
on if the upright position be assumed. Pericardial pain, rapid,
irregular heart, cough and dyspnœa coming on during the
course of rheumatism, nephritis, or general or local tubercu-
losis should direct our attention towards the pericardium.
When delirium occurs during an attack of acute rheumatism,
or coma or convulsions during the course of nephritis, you
should at once examine the heart. These symptoms are, in
these connections, especially indicative of danger if the patient
be a child.

While we may, by carefully noting the subjective signs
of pericarditis, suspect its presence, it is only by careful physi-
cal examination that we are able to diagnose it.

SYMPTOMS AND DIAGNOSIS.—The symptoms of pericar-
ditis will vary with the nature and extent of the inflammatory
process.

Clinically we encounter three classes of cases:

The first class comprises the acute form with plastic ex-
udation, including all cases from the "dry" pericarditis with
little or no exudation, to those extensive fibrinous exudates
which cause permanent thickening of the pericardium and
may result in adherent pericardium.

The second class comprises all grades of effusion, serous,
sero-fibrinous and purulent.

The third class comprises chronic pericarditis, or adherent
pericardium.

Acute fibrinous pericarditis can never be more than sus-
pected from the subjective signs, for the pain, disturbed
cardiac action and dyspnœa might equally apply to a localized
pleurisy. Inspection and palpation will tell alike of rapid,
forcible, irritable cardiac action which may be, but usually is
not, irregular. Palpation may also determine a friction
fremitus in exceptional instances. When present this is best
felt at the base of the heart in the third or fourth space, from
one-half to three-fourths of an inch to the left of the sternal
border.

The area of cardiac dullness remains unchanged unless
modified by some concomitant condition of the heart itself,
which is quite likely to be the case.

Auscultation gives us the most reliable sign of the disease —the pericardial friction sound. This sound is not always present, nor is it constant in position or character in the individual case. Depending upon the friction of surfaces covered with exudation it is liable to change place or character, or disappear with change in the character of the exudation. It may be ephemeral in character, lasting only a few hours, especially in those cases where effusion follows rapidly upon a moderate fibrinous exudation. Again the friction sound may last for several days. Friction sounds may be well defined during life and autopsy may show no exudation (Walsh: Pericarditis sicca). Hayden doubts, though does not deny, the possibility of friction sound without exudation.

Friction sounds may be heard over any portion of the cardiac area, but are most often found at the base of the heart about the junction of the fourth left rib with the sternum, or over the apex of the right ventricle between the fifth and sixth ribs just inside the left parasternal line. In some cases they may be heard behind, slightly above, and inside of the angle of the left scapula, even though scarcely audible in front (Sears).

The friction sounds may be single or double, systolic or diastolic or both, or may be independent of the heart sounds. In character they are grazing, rubbing or creaking. They are harsh in quality and the superficial origin of the sound is sometimes quite apparent, and again it is not easily determined. Pericardial friction sounds are louder when the patient is leaning forwards, or is in the recumbent position, than when standing or sitting. Friction sounds at the base may be louder at the end of a full inspiration, while those at the apex may be louder at the end of a complete expiration. Pleuritic friction sounds over the pericardium will cease with cessation of respiration. The so-called pleuro-pericardial friction sounds may continue during cessation of respiration; they have a fixed area of maximum intensity, while pericardial sounds are variable in this respect.

In like manner the intensity of exo-pericardial murmurs is the more constant. The location of pleuro-pericardial sounds is at the border of the pericardium and they may be

transmitted beyond the border, while pericardial sounds alone
are confined to the cardiac area.

Walsh mentions a murmur from pulsation of the aorta
against the pleura; it is diastolic in rhythm and is increased
by a full inspiration.

We have here a patient exhibiting the usual features of a
case of pericarditis with moderate fibrinous exudation. The
history is briefly as follows:

Female, age 17. Three months ago had an attack of
acute rheumatism involving ankles and left hand. Was treated
by family physician. Rheumatism disappeared. Six weeks
ago had another slight attack which also responded to treat-
ment. Two weeks ago rheumatism returned in left hand
only. She was weak and had disturbed heart's action. For
the last four or five days has had pain in cardiac region, palpi-
tation and slight dyspnœa on exertion. Her temperature is
100.5° F. Pulse 120, slightly remittent, quick, jerky and
irritable. You notice that there is increased force and slightly
increased area of apex impulse; a forcible, jerky motion which
increases decidedly with slight exertion or excitement. On
auscultation you hear at the base of the heart a to-and-fro
sound rather harsh in character, short in time, the systolic part
of the sound being longer and more distinct than the diastolic.
The sounds are synchronous with the heart sounds. They
have a distinctly superficial character and are increased by
leaning the patient forward. They are not so loud when the
patient lies down. They are not heard behind. At the apex
there is a murmur indicative of mitral stenosis which illus-
trates the frequent association of pericardial and endocardial
inflammations already mentioned. In differentiating the en-
docardial from the pericardial sound in this case we note that
the pericardial sound is superficial in character and rendered
more so by a change in the position of the patient. It is double
and not necessarily synchronous with the heart sounds. It
has a limited area of diffusion and no special direction of
transmission.

Pericarditis with Effusion.—In our second clinical divis-

ion we include all cases showing effusion into the pericardial
sac. Whether the fluid is purulent or not cannot always be
told from the symptoms. It may be suspected from the eti-
ological relations that the effusion is purulent, but it can only
be proven by inspection of the fluid.

The symptoms of effusion into the pericardial sac will
vary with the amount of the effusion. Small effusions are
difficult or impossible to make out and even in large effusions
the diagnosis may, at times, present many difficulties. The
question of surgical treatment renders the diagnosis of peri-
cardial effusion doubly important. Special signs are given
as diagnostic of small effusions, such as dullness in the right,
fifth interspace, the contrary condition—resonance in this situ-
ation—being indicative of the absence of effusion (Rotch).
The diagnosis of a small effusion is always a matter of doubt.
The practical necessity is for us to be able to diagnose effusions
which are large enough to give reliable symptoms, and which
are apt to be dangerous, and that present the necessity for
interference. Large effusions in uncomplicated cases can
usually be diagnosed without much difficulty. In case of con-
siderable effusion inspection will show a loss of respiratory
motion over the præcordium. There may be some extra
prominence or bulging of the præcordial region in young chil-
dren. The area of cardiac motion is indistinct and the apex
beat cannot be seen. So far as inspection is concerned we may
only surmise the nature of the trouble. By palpation we may
feel a wavy, undulating motion in large effusions; the impulse
of the heart against the chest wall will be very feeble compared
with its apparent effort. The apex beat is raised and carried
to the left. The character of the impulse gives the sensation
of imperfect contact with the chest wall. The elevation of the
apex is due to a forcing of the apex away from the chest wall
and bringing the side of the ventricle into contact with the
chest.

Percussion will furnish us the most decisive evidence of
effusion. The modification of the normal area of cardiac dull-
ness will vary with the condition of the heart, lungs and
pleura. If there is no special resistance to distention of the
pericardial sac the effusion will distend it in all directions,

with, perhaps, somewhat greater increase in the lateral dimensions than in the vertical. This is more apparent at the level of the fifth or sixth ribs than above. The outline of flatness is said to be a blunt cone extending from the first or second rib to the sixth or seventh, and from one inch or more to the right of the right sternal border to a varying distance to the left of the left mammillary line.

Various observers differ somewhat in regard to the area of flatness. Rotch claimed to have demonstrated by injections into the pericardial sac a much greater increase in the lateral diameter of the flatness than in the vertical. Bruen thinks that clinically we do not find great disproportion between the lateral and vertical increase in flatness. Shattuck believes that the flatness increases nearly equally in all directions. I believe that Rotch places too much stress upon the increase in the lateral area of dullness, and that practically the general increase in flatness in all directions is the important point to demonstrate.

Two very important points are the relation of the left line of dullness to the apparent apex beat; and the abrupt transition from flatness to pulmonary resonance at the left border of the area of flatness.

The extension of dullness beyond the apex beat is a most indicative sign of pericardial effusion.

The extension of dullness below the apparent apex beat is of the utmost importance in differentiating cardiac enlargement from effusion.

A distinctly pyramidal or triangular form of dullness is due to the nature of the displacement of the edges of the lung, and not to the shape of the pericardial sac.

Variations in the area of dullness from day to day caused by a varying amount of effusion is important, as is also variations in the outline of dullness upon change in position of the patient while the apex maintains the same relative position to the chest wall. The difference in the sense of resistance offered to the fingers as a pleximeter over the effusion and over the heart itself is important.

Ewart gives the following signs as the most important in an effusion large enough to bring up the question of sur-

gical interference: Considerable increase in the total area of dullness, as shown by the lateral boundaries; great increase in the area of absolute dullness; absolute dullness over
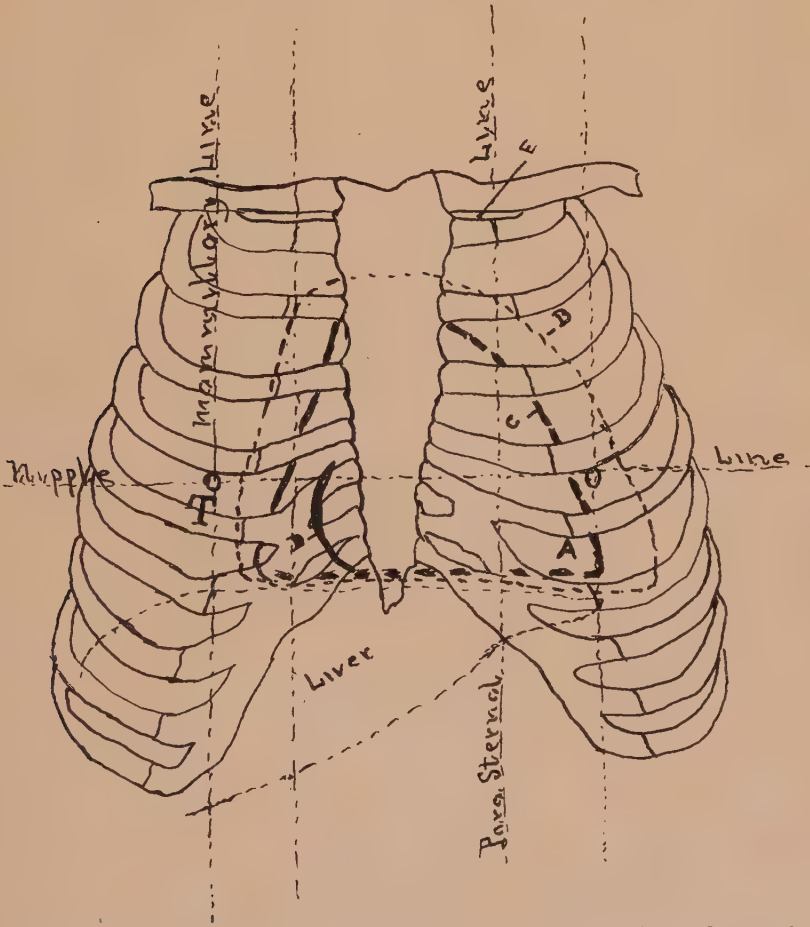
Diagram of physical signs of pericarditis. A.—Apex beat *above* and *inside* lower left angle of dullness. B.—Outline of relative dullness. C.—Outline of actual dullness. D.—Relative angles of dullness in dilatation (inner angle) and effusion (outer angle). Rotch's sign. T.—Inframammary patch of tubular breathing. E.—Position of first rib sign.

sternum; depression of the liver; dullness in the right fifth inter-cartilaginous space (Rotch's sign); alteration in the lower angles of dullness as distinguishing pericardial effusion

from cardiac dilatation—the right lower angle projects obliquely towards the right effusion—the left lower angle of dullness is a prominent angle in effusion and the apex beat is heard inside and above this angle; ability to feel the upper edge of the first rib as far as its sternal attachment, probably due to raising of the clavicle and relaxation of the sterno-costal ligament (this sign may possibly be found in some cases of cardiac enlargement) : a posterior patch of dullness at the base of the left lung extending from the spine outwards about as far as the line of the angle of the scapula, with an abrupt vertical outer
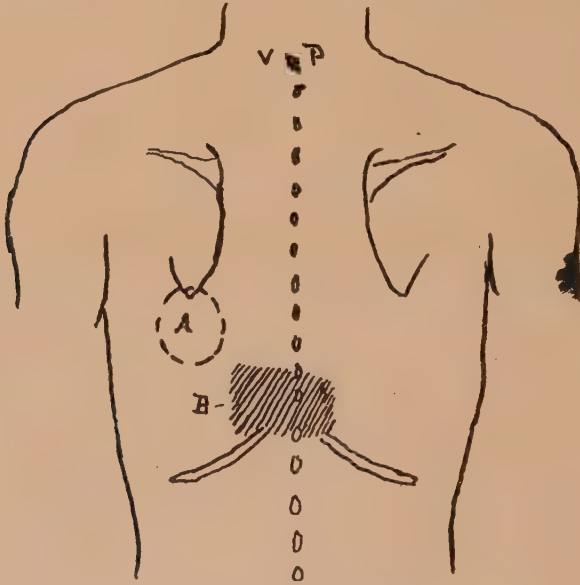
B.—Posterior pericardial patch of dullness.   A.—Posterior pericardial patch of tubular breathing (after Ewart).

boundary (this dullness may extend as high as the level of the ninth or tenth rib, and this boundary is also abrupt); an area of tubular breathing below the right mamma in the nipple line a little above the hepatic line (this tubular breathing may be restricted to expiration and is not constant); a posterior patch of tubular breathing and egophony about two inches in diameter immediately below or slightly to the left of the tip of the left scapula; secondary pleural effusions; a large slapping pulse much resembling the pulse of aortic regurgitation.

The posterior patch of dullness with abrupt boundaries is a valuable sign as it is not apt to occur in other conditions than pericardial effusion. The posterior patch of tubular breathing and egophony may be present frequently in pleurisy.

Tubular breathing beneath the right mamma is in my experience very inconstant. I have never been able to determine the first rib sign with any certainty.

Mediastinal tumors, pleurisy localized in the pericardial area and cardiac dilatation, are most likely to confuse us in making a diagnosis. The clinical history of these conditions differs from that of effusion.

Tumors have an irregular outline of dullness. Localized pleurisy will not present the angles of dullness of a pericardial effusion, nor will there be such abrupt transition from dullness to lung resonance. In cardiac dilatation the lesser degree of absolute dullness, the absence of dullness in the fifth right inter-cartilaginous space, the more gradual shading of dullness into resonance and the identity of the apex beat with the lower left angle of dullness will usually enable us to decide the question when taken in connection with the history and other evidences of chronic cardiac disease. The following case illustrates these points:

Man aged 25; seen in consultation. Had had periodical attacks of rheumatism for several years. Ailing for several months. Present attack began five weeks ago with pain in region of heart and cardiac disturbance. In bed for last two weeks. Much worse in last three days. Area of cardiac dullness increased so much during last five days that attending physician suspected pericardial effusion. Percussion showed upper border of dullness at upper border of third rib; right border of dullness at level of fourth rib, one-half inch to right of right sternal border; left border of dullness one inch outside of left mammillary line at level of fourth rib. Apex below seventh rib one inch outside mammillary line. There was marked aortic regurgitation with relative mitral regurgitation murmurs. Intensification of second pulmonic sound, Corrigan pulse. The dullness did not extend beyond or below the position of the apex beat, the latter fixing the lower left angle of

dullness. No dullness in right, fifth space. Left border of dullness was not abrupt.

Diagnosis: Hypertrophic dilatation of left side from aortic regurgitation. Secondary mitral regurgitation. Dilatation of right ventricle.

Death four days afterwards.

In this case close observation would undoubtedly have shown that the rapid increase in the area of dullness was coincident with the development of increased pressure on the pulmonary valve and the signs of right ventricular failure.

Chronic Adhesive Pericarditis.—This variety of pericardial inflammation is more frequent than is generally supposed.

Broadbent found thirty-one cases of adherent pericardium in eighty-six cases of death from heart disease.

The diagnosis is difficult and often impossible, the symptoms not being in any way characteristic; they simply pertain to enlargement of the heart, cardiac failure in general and right ventricular failure in particular. There may be dyspnœa, tenderness over the liver, vomiting, œdema, ascites, pain over the præcordial area or irregular heart's action; all pertaining to cardiac failure.

The pulse may be irregular. The pulsus inspiratione intermittens, (*pulsus paradoxus*) has generally been considered as indicative of pericarditis. Kussmaul believes it is of diagnostic importance in indurated mediastino-pericarditis, and is due to fibrous cords dragging on the aorta during inspiration.

This form of pulse is characterized by disappearance of the radial pulse during inspiration. Kussmaul regarded this pulse, together with simultaneous swelling of the great veins of the neck, as characteristic of pericarditis with adhesions. The pulsus paradoxus may occur in the absence of intrathoracic disease (Summerbrodt). It is to be regarded as a sign of mediastinal disease and is not characteristic of pericarditis. (Harris).

Systolic depression of the third, fourth or fifth interspaces was regarded by Skoda as characteristic of adherent pericardium. It has been found, however, in cases where no

adhesions were present (Friedreich, Traube) and may occur where the heart is considerably enlarged and the anterior border of the lung displaced, where the lung is adherent to the parietal pleura or where the lung is indurated or collapsed. It is to be regarded as a valuable sign of adherent pericardium where other causes for the recession can be excluded. Systolic retraction of the lateral or posterior portion of the thoracic walls, generally only on the left side though occasionally on the right, is a reliable sign when obtained. It is due to traction of an adherent pericardium on the diaphragmatic attachments.

If the pericardium is adherent to the chest wall and the diaphragm there is interference with the descent of the diaphragm, as shown by the loss of motion of the upper abdominal wall in the left sub-costal angle.

Systolic recession of the site of the apex beat may be an important sign but may be present in other conditions. I have observed systolic recession of the site of the apex beat in several cases of valvular disease where it was improbable that there were adhesions.

The position of the apex beat may be changed by adhesions, or the apex beat may be fixed in a certain position and remain unchanged by any position of the patient or by inspiration or expiration—a most important sign.

A diastolic shock may be felt with the flat of the hand over the cardiac region.

There is nothing characteristic about the heart sounds. There may be reduplication of the second sound at the base, and the pulmonic second sound may or may not be accentuated, according as there is, or is not, increased tension in the pulmonary system.

Broadbent points out that a weak pulmonary second sound with an enlarged right ventricle must be regarded as presumptive evidence of pericardial adhesions. He lays stress on the importance of right ventricular failure when the physical signs are apparently not definite enough to account for such a condition. The right ventricular wall being thinner suffers more from the effects of pericardial disease than the left.

The following history illustrates the ordinary conditions of adherent pericardium:

Man, aged 43. Rheumatic history. Came under observation for cardiac disturbance. Examination showed aortic regurgitation with consecutive hypertrophic dilatation of left ventricle. There appeared to be more enlargement of the right ventricle than could be accounted for at this time because of the aortic lesion, as backward pressure through the mitral opening had not yet occurred. There was systolic retraction in the fifth space one and one-half inches from the sternal border and systolic recession of the site of the apex beat one and one-half inches farther to the left in the sixth space. There were no other signs of adherent pericardium.

Considerable improvement under treatment. About two years afterward cardiac disturbance more marked. Mitral regurgitant murmur. Extensive dilatation of right ventricle. Dropsy. Purpuric eruptions over trunk and lower limbs. Death.

Autopsy showed incompetent aortic valve, hypertrophic dilatation of left ventricle, marked enlargement of right ventricle with greater degree of hypertrophy than usually occurs when the right ventricle fails secondarily to left side failure.

The pericardium was everywhere adherent to the heart and the covering of the heart was torn in separating the parietal layer from the heart.

TREATMENT.—The therapeutic management of pericarditis must be instituted with a thorough appreciation of its secondary nature. In some cases you will regard the occurrence of pericarditis as an accident merely, in the course of some disease, which is to be managed with as little departure from the therapeutic measures already in use as possible. Again the activity of the process may make it the dominant feature of the case for the time being.

In acute pericarditis with plastic exudation various means have been recommended with a view of limiting the amount of exudation and the liability of effusion, also the formation of adhesions. Antiphlogistic measures have generally been abandoned as unsuited to a secondary affection. Leeches and blisters over the præcordium are still recommended by some. In sthenic cases bleeding may have some beneficial effect, but this can also be gained by milder measures.

I have never been satisfied of the good effect of blisters in limiting the exudation as claimed by some observers. They annoy the patient and disturb the heart's action and I do not like to place even small blisters over the præcordium.

Ice bags or coils to the præcordium will certainly give relief in many cases. Personally I have preferred the use of hot applications, especially in children in whom there is less danger of depression following the shock of hot than cold applications to the chest wall. Frequent, one-half-grain doses of calomel, combined with a little opium, are recommended to limit the extent of the exudation. I have never seen any evidence to convince me that calomel had this effect.

It is claimed that large doses of salicylate of soda will limit the extent and severity of the inflammation. While this is a difficult matter to decide, I do not see any evidence in the statistics available which proves this point, and my personal experience is against it.

The management of an attack of acute plastic pericarditis should be about as follows: If there is pain, hot applications to the præcordium. With a rapid, full pulse, fifteen grains of sodium bromide, with one or two drops of tincture of aconite, every four hours to quiet the circulation. Absolute rest in bed and isolation from all disturbing influences. Morphia or codeine should be used to quiet severe pain and restlessness; opium should be used with care however. Chloral, chloralamid, trional and similar drugs are unreliable and unsatisfactory. Moderate cholagogue catharsis should be maintained.

Pericarditis occurring with rheumatism or Bright's disease emphasizes the necessity of bringing these diseases more quickly and fully under the influence of therapeutic measures.

The Treatment of Pericardial Effusions.—The medicinal treatment of effusions is not effective. The use of diuretics, diaphoretics and cathartics is of little or no avail in removing effusions. When the effusion is sufficient to cause dangerous symptoms it is not safe to waste time on such measures. Stimulant and supportive treatment is of course indicated.

The first question is—Shall we interfere with a pericardial effusion? In deciding this we must first consider the primary cause. The well known tendency of rheumatic ef-

fusions to subside, even more quickly than they appear, will at times justify a delay that would be dangerous under other circumstances. In general; dyspnœa, tumultuous heart's action, irregular, feeble, dicrotic pulse, may be taken as sufficient warrant for puncture, especially in relation to pneumonia and nephritis as primary causes. I have, however, seen these symptoms well marked in rheumatic effusion, and while holding myself ready to puncture have had the effusion diminish within twelve hours sufficiently to render the patient quite comfortable. When the effusion reaches sufficient size to cause such symptoms, and the border of dullness remains the same for twelve or twenty-four hours, puncture is indicated.

The simplest instrument is the best for puncture. For serous effusions an ordinary aspirating needle may be used. If the effusion is large there is little danger of injury to the heart if the needle is handled carefully. A needle with a sliding point is best. A small trocar and canula attached to a rubber tube may be used as a siphon, or Pepper's double canula may be used. Roberts uses a trocar protected by a flexible tube.

The point for puncture is a matter for selection in every case. Successful puncture has been made in various situations. Roberts says to tap in the fossa between the ensiform cartilage and the costal cartilage on the left side; or in the fifth left interspace near the juncture of the sixth rib with its cartilage, or from two to two-and-a-quarter inches from the left border of the sternum. He favors the latter point most.

We must remember the internal mammary artery which descends to the lower border of the sixth costal cartilage. It is situated about four or five millimetres from the border of the sternum (Cruveilhier, Sappey), or according to Roberts about six to twelve millimetres from the sternal border. From the point of bifurcation the superior epigastric artery continues outward and downward in the sixth space.

Rotch advises puncture in the fifth right interspace at the sternal border. Shattuck has made his most successful punctures just inside of the left lateral border of dullness from one to two inches outside of the nipple line. He strongly favors the apex of the left costo-xiphoid angle for puncture. At

whichever point selected the instrument should be entered slowly and the vacuum turned on as soon as the needle is well engaged in the tissues, as the first intimation of the needle being in the sac may be the presence of fluid in the tube. There is no danger of removing too much fluid, but it is not wise to change the position of the needle point too often in hope of getting more.

The removal of a small portion of a serous effusion will contribute greatly to the comfort of the patient and facilitate the absorption of the remaining fluid.

My own preference in regard to the particular place to puncture is in the fifth interspace two inches from the left sternal border. In some cases of large effusion I would be in favor of the costo-xiphoid space, though I have never tapped in this situation. For purulent effusions continuous siphonage is possible but more uncertain than in purulent pleurisy. Free incision of the pericardial sac with or without drainage, should be made. The necessity of drainage will depend on the nature of the case; it does not appear to be necessary in all instances.

Irrigation of the sac is not advisable except in selected cases. Porter has shown by dissections that the point for incision is in the fifth left interspace at the sternal border.

The treatment of adherent pericardium resolves itself into maintaining the integrity of the heart, sustaining the general nutrition and teaching the patient his individual limitations relative to exercise, excitement and all disturbing influences; in other words the usual management of an incompetent heart.

The great majority of cases of adherent pericardium are serious in nature, especially when there is adhesion to the chest wall. It is probable that serious results fail to ensue only in those cases where adhesion to the diaphragm or chest wall has not occurred, and the heart is about normal in size.

We may expect somewhat better results in children in relation to the nature of the symptoms, than in adults, for a moderate degree of adhesion in a child will give much more obvious symptoms on account of the yielding nature of the chest wall.

As pericarditis is much more frequent in children than in adults, and is usually more readily diagnosed, we may hope for somewhat better results in treatment.

One of the greatest difficulties, however, in children is the impossibility of obtaining rest and quietude. Children accommodate themselves readily to altered circumstances and may readily overtax the heart without giving any subjective evidence of the fact until serious damage has occurred.

The outlook for cases of adherent pericardium is not, according to Broadbent, as discouraging as Corvisart, Hope and others would have us believe. Broadbent cites cases proving the long existence of adherent pericardium without any serious result.

The principal objects to be attained in the treatment of adherent pericardium are to sustain the nutrition of the heart and prevent over-exertion. Iron, strychnia and the iodides are useful, particularly strychnia.

Cardiac stimulants, including digitalis, should not be used unless under the necessity of dynamical failure.

To judge aright the capabilities of the heart and the exact limitations in the way of exercise which apply to individual cases, is very difficult. The conclusion, however, is forced upon us that, in adherent pericardium, treatment is followed by good results only in those cases where the more unfavorable conditions are absent.

PNEUMOPERICARDIUM.

Pneumopericardium, or air in the pericardial sac, is a rare affection. It was observed by Voegtel, and was described by Laennec. Schrötter, during a very extensive hospital experience, never saw a case. Skoda never had an opportunity to observe a case.

ÆTIOLOGY.—Pneumopericardium occurs from the extension of morbid processes in adjacent tissues whereby a fistulous communication is established with the pericardial sac. In this manner it has occurred from pyopneumothorax (Eisenlohr), from a pulmonary cavity (Stokes), from perforation of the diaphragm by a gastric ulcer (Säxinger), from cancer of the œsophagus (Begbie), and from hepatic abscess perforating stomach and pericardium (Graves). Again, pneumoperi-

cardium may result from traumatism, such as gunshot wounds, wounds from sharp instruments or from fractured ribs. The possibility of the spontaneous development of pneumopericardium has always had some adherents, and much discussion has taken place on this subject. The possibility of errors in diagnosis is, of course, great, and therefore it is difficult to say in any case that the spontaneous development of gas has taken place. The recent discovery by Welch of the bacillus aërogenes capsulatus, and the observations of Nuttall and Flexner which show that this organism causes subcutaneous emphysema in connection with wounds, and that the gas may be found in the blood, show that in connection with traumatic causes the spontaneous occurrence of gas in the pericardial sac is possible.

MORBID ANATOMY.—Pneumopericardium is attended by pericarditis, and there is exudation of either a hæmorrhagic, fibrinous, or purulent character. There will be yellowish, brownish, or red, fetid fluid in the pericardial sac. There may or may not be distention of the sac, depending on whether or not there is a communicating opening.

CLINICAL HISTORY.—Aside from a history of traumatism which would lead us in the proper direction, there is usually a history of chills, of high, fluctuating temperature with profuse perspiration during the remissions of fever. Insomnia and delirium are features. Dyspnœa is usually more or less marked. Cyanosis and œdema may be present. Palpitation and syncope may have occurred. Severe pains over the front of the chest may be complained of. Usually pneumopericardium is fatal in from a few hours to three or four days. It is almost invariably fatal when fistula is a cause. When traumatic it has been known to recover. The gas may disappear very rapidly.

SYMPTOMS AND DIAGNOSIS.—The pulse varies in rate and is empty, irregular, and very compressible. There may be slight bulging of the præcordial region, though this is not the rule. The visible impulse of the heart has disappeared, nor can it be felt. Percussion of the præcordial area gives a tympanitic note which changes in character with change in the position of the patient. According to Stokes it is possible to

obtain a cracked-pot sound on percussion. With the patient recumbent the entire præcordial area may be tympanitic. With the patient sitting the lower portion of the area is dull from gravitation of the fluid in the sac.

On auscultation we may hear splashing noises which have been likened to those produced by a churn or by a water-wheel. As these sounds are synchronous with the heart movements, they are regular or irregular according as the heart is rhythmic or arrhythmic. These sounds are metallic in character, and may be echo-like. There may be friction sounds or endocardial murmurs. At times the splashing sounds may be heard at some distance from the chest wall.

The diagnosis is usually not difficult and rests on the splashing metallic sounds heard on auscultation. A dilated stomach may cause confusion as it may give tympany in the lower præcordial region, and splashing from transmitted cardiac motion, but the latter disappears on assuming the erect position, while the upper præcordium is not tympanitic.

TREATMENT.—When the disease arises from the extension of adjacent morbid processes the treatment is palliative only. In traumatic cases strict antiseptic measures must be adopted while the heart is carefully watched and stimulated as necessary. If there is pericardial effusion, especially if it be purulent, its removal may be necessary.

HYDROPERICARDIUM.

Usually after death the pericardial sac contains from 5 to 10 c. cm. of yellowish liquor pericardii. The quantity may be as much as from 90 to 100 c. cm., but much above this quantity is probably pathological. Pericardial dropsy consists of a serous transudation into the pericardial sac. It is analogous to hydrothorax.

ÆTIOLOGY.—The affection is always secondary. It arises from the circulatory conditions which develop in valvular or myocardial disease, or in connection with disease of the lungs, pleuræ or kidneys. New formations in the heart, pericardium, or mediastinum may include hydropericardium among their

pressure effects. Various cachexias may cause hydropericardium.

MORBID ANATOMY.—The fluid is clear or of a yellowish or greenish tint with a dichroic tendency. It may be red in color through the presence of blood, or possibly brownish in color. Its sp. gr. is about 1015. The quantity of fluid may be about 1,000 c. c. or even as much as 4,000 c. c. (Corvisart). It may contain epithelial or round granular cells, small fibrinous masses, and possibly cholesterine crystals. The pericardial sac will be more or less distended and may give fluctuation. The serous surfaces are smooth and shining and pale. The pericardium may be thin, or, in slowly developed cases, thick. The sub-epicardial fat may have disappeared, and the adjacent tissues may be œdematous. The tissue of the heart is pale and flabby and the right auricle and ventricle may be considerably dilated.

CLINICAL HISTORY.—The clinical history of this condition will be mainly that of its associated diseases. It is usually the terminal event in a series of pathological states. There may or may not be fever. There may be subnormal temperature. Dyspnœa, cyanosis, syncope, oppression in the chest, œdema, somnolence, delirium, and nervous symptoms are features.

SYMPTOMS AND DIAGNOSIS.—The patient assumes a sitting posture, leaning forward with arms on knees. The pulse is small, compressible, frequent and irregular. The cervical veins are full. The præcordium may be prominent and the corresponding intercostal spaces widened. The cardiac impulse is absent, vocal fremitus over the præcordium is diminished. The area of cardiac dullness is increased and of the same shape as in other exudations. The heart sounds are feeble. There is no friction sound. The liver may be enlarged, and there may be ascites. The urine is diminished in quantity and contains albumen. There may be œdema of the lungs or hydrothorax.

The diagnosis rests on the signs of effusion and the absence of any proof of pericardial inflammation, also on the presence of conditions likely to cause hydropericardium.

TREATMENT.—The treatment is that applicable to the

cause of the disease. Dry food and the use of diuretics and purgatives are indicated. Paracentesis may be indicated.

HÆMOPERICARDIUM.

Those forms of pericardial inflammation such as occur with scurvy, cancer, tubercle, kidney disease, etc., and which may be attended with bloody exudation, do not belong under this heading. The pathological conditions and clinical features of hæmopericardium are entirely different from those of hæmorrhagic pericarditis.

Hæmopericardium arises from direct or indirect injury, as from blows, falls, injuries by projectiles or sharp instruments. It also occurs from pathological causes such as rupture of the heart when diseased and under a strain, rupture of a coronary artery the seat of marked degeneration, rupture of the aorta, or of an aneurism.

The amount of blood in the pericardial sac may be small or considerable. In rapidly developed cases it is usually small, while in slowly developed cases it may be considerable. The blood may be coagulated or fluid, but there is usually only partial coagulation.

The clinical features depend on the nature of the cause. In rapidly developed cases the patient may suddenly pass from apparent good health into a pulseless collapse. In slowly developed cases there is more gradual development of an asystolic condition of the heart. There may be pain in the præcordium, or a sensation of something breaking in the region of the heart followed by dizziness or syncope. The skin is pallid, with cold perspiration. The pulse is feeble, irregular, or may be imperceptible. Physical signs are absent, except possibly in slowly developed cases where there may be signs of increased area of cardiac dullness. Sudden collapse and rapidly acquired enlargement of the area of cardiac dullness in connection with some disease likely to result in hæmopericardium will establish the diagnosis.

The treatment, if there be any opportunity, must be surgical. The prospects of surgical treatment of the heart and

pericardium is much better than has been supposed. Minni finds that only 19 per cent. of penetrating wounds of the heart are immediately fatal. Elsberg, Podrez, and others consider the suturing of wounds of the heart as justifiable and, under certain conditions, successful. New operations have been devised by Cyril Ogle and by E. Rotter with which to gain access to the heart, but they have yet to be demonstrated on the human subject.

PERICARDIAL TUBERCULOSIS.

Tubercular affections of the pericardium were recognized by Corvisart and by Laennec. They are usually of secondary occurrence either from the extension of tubercular processes or as part of a general infection. Pericardial tuberculosis may in rare instances occur primarily, as was pointed out by Virchow. Simple pericarditis occurring in a tuberculous subject is not uncommon, but this form should not be confounded with tubercular pericarditis.

ÆTIOLOGY.—The affection occurs at any age, but is most common between 15 and 30. Cases have been reported in a child of eight months, and in a woman of 88 years. According to Osler it is more common in men than in women. Primary cases in very young children occur either through obscure infection in subjects with marked heredity, or through latent congenital tuberculosis. Secondary cases are usually associated with tuberculosis of the pleura, lung or bronchial glands, especially the latter. In acute general tuberculosis we have the pericardium involved only when there is involvement of the other serous membranes (Osler)—a serous membrane tuberculosis (Strümpell).

MORBID ANATOMY.—Tubercular inflammations of the pericardium are similar to those of the pleura. They may be associated with serous or fibrinous exudate. Small nodules are found scattered along the course of the cardiac vessels. Osler says there are two classes of cases; one in which there are firm adhesions with great thickening of the pericardium, and one in which there is recent exudation of fibrinous, sero-

fibrinous, hæmorrhagic or purulent material. The former is most frequent. In some instances of tubercular pericarditis there may be no tubercles, though bacilli be present (Kast). There may be marked tendency to hæmorrhage. Ulceration of the pericardium or epicardium may occur. The heart muscle may even rupture from this cause (Eichhorst).

CLINICAL HISTORY.—The course of the disease may be so latent that it is only discovered after death. Usually there are the ordinary symptoms of tuberculosis, in a greater or less degree.

SYMPTOMS AND DIAGNOSIS.—The local symptoms are obscure and not well marked. When present there are the usual signs of pericarditis. Chronicity is the chief feature of both clinical history and symptoms. The diagnosis rests on the evidences of pericarditis together with the general conditions of tuberculosis, and, perhaps, local tuberculosis in some tissue.

TREATMENT.—The treatment is conducted along general lines, with special attention to the integrity of the heart. If exudation is excessive we may have to resort to aspiration or to incision, the former preferred.

PERICARDIAL SYPHILIS.

Syphilitic pericarditis is rare. Usually it occurs as a late secondary or tertiary manifestation, but may be congenital. Gummata are rare. More often there is fibrous infiltration leading to adhesion of the epicardium and pericardium. Obliteration of the sac is usually not attained. There may be fluid exudation.

There are no characteristic symptoms. The affection is usually not diagnosed during life. It may be suspected when evidences of indurative pericarditis present in persons with systemic infection of specific nature.

The treatment consists of the use of iodide of potassium, mercury, and, perhaps, of some mercurial ointment applied to the præcordium.

PERICARDIAL NEOPLASMS.

Carcinoma and sarcoma of the pericardium are compara-tively rare. Primarily, they are very rare, secondarily they occur in connection with malignant disease of the mediastinal glands, lung, pleuræ, or œsophagus. Occasionally they may result from metastasis from a distant organ. The tissue changes will correspond to the nature of the growth plus some infiltration of the serous and subserous strictures. There will be some fluid in the sac of a hæmorrhagic, serous, or purulent nature. If there is much pressure there may be considerable transudation.

Actinomycosis of the pericardium may occur in connec-tion with like disease in other tissues. It is very rare (Münch). Hydatid cysts of the pericardium have been observed by Habershorn, Landousy and others. Small free bodies, or fringes similar to those found in the synovial cavities have been described by Bouchard as occurring in the pericardial sac.

New formations in the pericardium can only be sus-picioned when evidences of pericardial involvement are associ-ated with the presence of like disease in other organs. The treatment is, of course, merely tentative.

CHAPTER II.

MYOCARDITIS.

Myocarditis is a rare affection as compared with the frequency of other cardiac lesions.

Of late years myocarditis has not been so common a diagnosis as formerly. Many cases which were previously classed under myocarditis, belonged undoubtedly to the degenerations of the cardiac muscle. These two conditions are, however, so intimately blended, both in their ætiological and pathological relations that a clinical classification is not always possible, and even pathological classifications may be difficult, owing to the mixed nature of the tissue changes. Any serious degree of myocarditis can hardly occur without some accompanying features of degeneration, and if this be true in a pathological sense, the clinical manifestations must present an even greater ambiguity.

There are, however, cases in which both the pathological state and the clinical symptoms are more clearly defined, and in which a definite diagnosis may be arrived at.

Myocarditis may be acute or chronic. It affects the left heart more frequently than the right. Either of these forms may be circumscribed or diffuse.

Circumscribed myocarditis of either acute or chronic nature will occur in connection with other pathological states and is not to be diagnosed except under exceptional conditions. Diffuse myocarditis, either acute or chronic, is of direct clinical interest. The chronic form is much more frequent than the acute.

ÆTIOLOGY.—In association with other cardiac lesions myocarditis may be due to pericarditis, endocarditis or to sclerosis or embolism of the coronary arteries. Rheumatism may cause acute myocarditis independently of other cardiac

disease. Pyæmia and septicæmia may cause acute localized myocarditis (cardiac abscess).

Acute diffuse myocarditis may occur in connection with typhoid and typhus fever or acute ulcerative endocarditis.

Aufrecht claims that alcohol may cause myocarditis. The anatomical findings, however, by which he identifies the condition, are more those of a mixed sclerosis and degeneration, and the clinical picture is that of chronic degeneration of the heart muscle.

Chronic myocarditis may occur with all other cardiac lesions. The most frequent causes are rheumatism and nephritis. Syphilis may also cause myocarditis.

Chronic myocarditis may occur in connection with the vascular changes of senility. Arterial sclerosis, end-arteritis and peri-arteritis are frequently associated with chronic myocarditis.

MORBID ANATOMY.—In acute myocarditis secondary to pericarditis or endocarditis the tissues adjacent to the primary inflammation alone may be involved. The muscular tissue is infiltrated with small cells, the fibres are softened and may have undergone granular degeneration or coagulation necrosis.

In the more diffuse forms, such as may occur with rheumatism or with prolonged high temperature from any cause, the changes are more general but affect the left ventricular wall more than the right. The nuclei of the fibres are large and prominent. Leucocytes infiltrate the tissues, and the change may assume the nature of "cloudy swelling," an initial step in the process of fatty degeneration.

These changes affect severely the contractility of the muscle and explain why there is such sudden and marked cardiac asthenia in these cases.

In acute myocarditis from pyæmia, single or multiple abscesses may form in the cardiac wall; these may rupture into the cavities of the heart or into the pericardial space.

Chronic myocarditis is characterized by the development of fibrous tissue between the muscular elements of the heart wall. The intermuscular septa around the blood vessels is the primary seat of this process.

Disease of the coronary arteries is most frequently asso-

ciated with chronic myocarditis. Embolism or thrombosis of small branches of the coronary artery may cause the localized form.

When secondary to pericarditis, chronic myocarditis affects the external portion of the heart wall and both ventricles alike. When secondary to endocarditis it is the inner portion of the wall which is the most affected and the left side is most frequently involved.

There may be small or large, single or multiple patches of induration, or there may be a diffuse fibroid development.

The loss of contractility of a portion of the wall through the development of a fibroid area may result in a cardiac aneurism. The favorite seat for these pouches is the apex of the left ventricle and the upper portion of the interventricular septum. Rupture of the heart from this cause is very rare.

Syphilitic myocarditis occurs as grayish nodules or gummata, or as localized fibrosis, the latter not differing essentially from fibrous induration from other causes. General infarction may occur from the rupture of softened gummata into the heart cavities (Oppolzer).

More or less degeneration of the muscle fibre occurs in the region contiguous to areas of fibrosis, and the integrity of the heart is profoundly affected.

CLINICAL HISTORY.—The clinical history of myocarditis is unimportant. Neither the acute or chronic forms exhibit any history of value. A series of embolic accidents may, in the absence of endocarditis, be indicative of cardiac aneurism resulting from a patch of induration.

Pyæmic manifestations in connection with recently developed cardiac symptoms may indicate acute localized myocarditis (abscess).

Continuous high temperature in connection with any of the infectious diseases or with rheumatism may point toward acute diffuse myocarditis, where rapid loss of heart power occurs accompanied by dilation of the heart.

Chronic diffuse myocarditis presents the clinical history simply of ataxia of the heart common to other cardiopathies.

SYMPTOMS AND DIAGNOSIS.—*Acute circumscribed myocarditis* has no characteristic symptoms and cannot be diag-

nosed with certainty. Rapidly developed cardiac weakness, oppression, faintness, rigors, delirium, sudden appearance of a cardiac murmur, or sudden paralysis of the heart in connection with some infectious process, may indicate the nature of the trouble. Rühle says that the symptoms may be those of acute poisoning or of internal hæmorrhage.

Chronic circumscribed myocarditis can only be diagnosed when it results in thinning or pouching of the cardiac wall and the resultant aneurism is so situated as to be outlined by percussion or palpation. The changes that occur in the rhythm of the heart's action and in the nature of its sounds are of little diagnostic value as they cannot be distinguished from those which occur from degeneration of the muscle.

I think the statement that degeneration is less liable to exhibit cardiac enlargement than is aneurism (M. P. Jacobi) is misleading. It is very rare for true cardiac aneurism to exhibit sufficient cardiac enlargement to enable a diagnosis to be made *intra vitam*.

Acute diffuse myocarditis is rarely recognized during life. The rarity of its occurrence, together with symptoms of a character common to other cardiac diseases, explain this. A rapid, feeble, compressible, irregular pulse, appearing suddenly during the course of some affection which myocarditis is liable to complicate, is the most important symptom. There may be dyspnœa and cyanosis, restlessness and anxiety, or delirium. The heart at first labors violently and the sounds are short and sharp; later the cardiac action is weak and the sounds are feeble and indistinct.

There are no special physical signs. Dilatation of the heart is generally present to some extent but cannot be readily determined.

Chronic diffuse myocarditis is the most common variety of myocarditis, and from a clinical standpoint the most important, for, while the diagnosis is very difficult, it may be recognized early enough to render treatment of some avail. Rühle states that the clinical picture is that of a non-compensated valvular lesion, while Fraentzel says his cases have all exhibited failure of the lesser circulation as the first symptoms.

Exhaustion on exertion or excitement accompanied by

palpitation and præcordial pain are prominent symptoms. These attacks are, at first, quite transient, but later may become frequent and prolonged. They are likely to occur at night, especially attacks of cardiac asthma which resemble a true asthmatic paroxysm. The patient, however, will be much quieter than with an attack of bronchial asthma as the slightest movement increases his distress. The blood pressure during these attacks is high and in many instances the attacks are due to vascular contraction through excitement of the vaso-motor system. Transient pulmonary congestion and œdema may be present, but they are the result rather than the cause of the condition leading to the paroxysm.

The præcordial pain is irregular and of varying severity. The onset of the pain is not sudden and it may be merely a sensation of distress. The pain resembles the so-called pseudo-angina, or the *angina sine dolore*.

There may or may not be perceptible enlargement of the heart. The apex beat is weak and diffused. The pulse is rapid and irregular in both force and frequency. According to Rigal the pulse is regular, but in my experience, a rapid, irregular pulse which changes pace frequently without apparent cause, is one of the most characteristic features of this disease.

The heart rate may become so rapid as to constitute a *delirium cordis* (Rühle). Again, the pulse may be regular and of normal rate or even a condition of bradycardia may be present.

The character and quality of the heart sounds in chronic myocarditis are characteristic. The second sound is weak compared with the bulk or volume of the first sound, whereas in most other conditions of cardiac weakness the second sound is relatively louder than the normal. The variation in quality of the cardiac sounds is also characteristic. A few tones will be loud and snappy while the following sounds will be weak and indistinct,—a scarcely audible tic-tac. These sudden variations in the rhythm and character of the cardiac sounds are more marked in chronic diffuse myocarditis than in any other condition involving dynamical failure of the heart.

These symptoms appearing in connection with vascular

sclerosis or chronic nephritis,—conditions in which we may expect those concomitant changes in the arteries or myocardium which are the initial process leading to the diffuse myocarditis, are strongly characteristic of the latter affection. Later on we have passive congestions and dropsy from general failure of the circulation. These symptoms differ in no way from the same manifestations of cardiac failure from other causes.

The following history illustrates a case of chronic diffuse myocarditis:

Man aged forty-six, single, moderate drinker, had two or three attacks of rheumatism during the last ten years. Never confined to bed. Syphilitic history(?) Had severe attack of mountain fever one year ago. Subsequently suffered from præcordial pain, dyspnœa and insomnia; at times anorexia and vomiting. The dyspnœa and præcordial distress became more constant. At time of coming under observation was confined to house. Œdema of extremities, congestion of liver. Suffered much from gastric derangement. Pulse irregular, from 100 to 130; heart sounds vary much; second sound weak, first sound muffled and indistinct. Variation in rate of pulse sudden and frequent. The heart was enlarged from hypertrophic dilatation of left ventricle. Slight aortic stenotic murmur. The arteries were sclerotic and tension high. Improved under treatment for short time. Heart became more dilated and death occurred nine months after coming under observation.

Autopsy showed chronic endocarditis with aortic localization, hypertrophy and dilation of both sides, sclerosis of coronary and systemic arteries, chronic diffuse myocarditis with fatty degeneration of fibres of heart muscle. The left kidney very small and firmly contracted. Right kidney slightly larger than normal and, beyond slight parenchymatous changes, was healthy.

The clinical diagnosis of myocarditis is difficult and rests mainly on exclusion of other causes than myocarditis for the abnormal condition of the heart. It is most apt to be confounded with the earlier stages of degeneration of the heart muscle.

In degeneration of anæmic origin and in fatty infiltration the ætiological relations will aid the diagnosis. In the toxic degenerations, such as occur with parenchymatous nephritis, enlargement of the heart is more pronounced, for while slight hypertrophic enlargement is frequent in chronic myocarditis, it is never so prominent a diagnostic feature as the hypertrophic dilatation which is so conspicuous in many cases of parenchymatous nephritis.

The duration of myocarditis is variable. The acute form may last but a few days or weeks. The chronic form is of such insidious advent that its limitations are difficult to fix. The cases are usually well advanced before coming under observation, and their clinical history ranges from three or four months or two or three years.

TREATMENT.—The treatment of *acute myocarditis* is largely embodied in the management of the condition with which it is associated. Its occurrence brings a demand for stimulation and support to the heart. This must be active, as the demand is urgent and sudden. This indication is best met by alcohol, strychnia and ammonia.

When, in any condition with which acute myocarditis is likely to occur, we find the pulse becoming weaker, faster and irregular; the patient anxious and restless, we cannot wait for the action of digitalis. Moreover, digitalis is of questionable utility in these cases. Nitro-glycerine is not suitable and is dangerous. We must therefore rely on the remedies mentioned. Half-ounce to one ounce doses of whiskey containing one twentieth of a grain of strychnia may be given every two to four hours. Strychnia if used at all must be used in large doses for its stimulant effect.

One-half to one dram doses of the aromatic spirits of ammonia, or two to three dram doses of the liquor of the acetate of ammonia, may be used in place of, or in connection with, the alcohol.

Opium may be used very carefully to quiet the patient in case of great restlessness, as the disturbance incident to a delirious condition may be more dangerous than the amount of opium necessary to quiet the patient.

The patient must not be allowed to make a movement

that can be obviated, as the slightest exertion may cause syncope.

Chronic myocarditis must be treated, first, with special reference to its ætiological relations; second, with regard to the condition of the systemic arteries and blood pressure; third, with regard to the propulsive power of the heart itself. The main object is to improve nutrition and maintain elimination. If these processes are below normal then degeneration of the heart muscle fibres will progress rapidly and asystolism will soon develop. The elimination should be as closely studied as in nephritis, whether the latter condition be associated with the case or not.

If rheumatism or syphilis be the ætiological factor in the case, such relations to the disease should be kept in mind even though all manifestations of the basic conditions have vanished. If general sclerosis of the vessels be present then the continuous administration of vaso dilators is necessary, for very slight increase in intra-ventricular pressure will produce dilatation in a heart weakened by myocardial sclerosis. For this purpose nitrite of sodium (two or three grain doses), nitro-glycerine (one two-hundredth grain), or opium (two to five drops of the deodorized tincture), may be given three times daily. As to the heart itself, we must remember the great probability of imperfect coronary circulation, hence we give sodium iodide (five to seven grain doses) for its dilating action on the smaller arteries. Strychnia (one-thirtieth grain doses) should also be given. These drugs are suitable for all stages of the disease and should be continued for months. Digitalis is not well borne as a rule, but the weak, irregular heart, with præcordial pain is often much improved by strophanthus. Seven to ten drop doses of the tincture in aqua lauro-cerasi, combined with the doses of strychnia and iodide of sodium before mentioned, makes an efficient combination for these cases.

CARDIAC DEGENERATION.

Under the generic term *degeneration,* we include fatty infiltration, fatty degeneration (fatty metamorphosis), and granular degeneration (parenchymatous degeneration).

While these morbid processes are essentially different, suffi-
cient warrant is found for such a combined classification in
the pathological fact of the inter-dependence of these pro-
cesses and their mutual clinical association.

ÆTIOLOGY.—*Fatty infiltration* occurs in connection with
a general increase of bodily fat resulting from disturbance of
the proper relations existing between food supply and oxida-
tion. The factors in the production of this state are the exces-
sive ingestion of hydrocarbons and lack of proper exercise
with imperfect elimination. Fatty infiltration may occur sec-
ondary to fatty degeneration of the muscle fibre.

Fatty degeneration of the heart may be diffuse or circum-
scribed; it results from diminished blood supply, changes in
blood quality from high temperature or poisons, and is part of
the change incident to the decay of senility.

Fatty degeneration may be associated with all varieties
of cardiac lesions as a result of the various ways in which they
interfere with the nutrition of the heart muscle. Localized
fatty degeneration results from the various forms of coronary
obstruction, peri and endocarditis, and in all instances where
there is local interference with nutrition. It may be second-
ary to fatty infiltration and to the perivascular scleroses, both
arterial and venous, which result from aortic and mitral lesions
(Banti), in which connection it marks the beginning of actual
loss of compensation. Diffuse fatty degeneration occurs from
chlorosis, pernicious anæmia, tuberculosis, phosphorus and
arsenic poisoning, and in all instances where the quality and
quantity of the blood supplied the heart as a whole, is defective.

Granular degeneration, (parenchymatous degeneration).
This form of degeneration is due to toxicity of the blood which
may or may not be attended with high temperature. It occurs
in the acute diseases and is frequently the initial step in the
process which results in fatty degeneration.

Granular degeneration of the heart occurs from the auto-
intoxications resulting from nephritic and intestinal toxæmia.
Parenchymatous nephritis is particularly liable to be associated
with this form of cardiac degeneration. Senator expressed
this view indefinitely when he said that the hypertrophy of
parenchymatous nephritis was eccentric, implying a concomi-

tant degeneration of the muscle. The nature of the poisons resulting from renal insufficiency are very complex, and the associated cardiac lesions are correspondingly varied. The manner of development, however, of that cardiopathic state so frequently found associated with parenchymatous nephritis, indicates a muscular weakness which can only be explained by the effect on the heart muscle of toxic substances.

MORBID ANATOMY.—Fatty infiltration is characterized by a more or less regular deposit of fat between the fibres of the heart muscle. Normally there is some fat beneath the visceral layer of the pericardium. It is usually found along the grooves between the auricles and ventricles. The fat may increase so as to completely cover the right ventricle. The left ventricle is seldom or never completely covered. The fat collects along the vessels and between the muscular fibres. It is more plentiful in the outer wall than towards the endocardium. In moderate infiltration there are irregular collections of fat, while in advanced cases, the whole ventricular wall may have the appearance of a fatty layer, the muscular tissue being discernible only by the microscope.

The presence of fat between the muscular fibres interferes with nutrition and results in fatty degeneration of the fibres.

Fatty degeneration.—This form of degeneration is due to a metamorphosis of the protoplasm of the cells, and is, in a measure, a perversion of the normal physiological processes of cell life. The protoplasm of the cells is changed into molecular fat showing as minute granules, first in the protoplasm then in the nucleus.

This form of degeneration is met with in the heart more frequently than in other organs. It is associated, to a greater or less degree, with the advanced stages of nearly all cardiac lesions. It may terminate in absorption, caseation, calcification or softening. The advanced stages are only met with in localized forms of the disease.

The heart muscle is soft, pale and friable. The fibres lose their striation and contain granules of fat. According to Walsh the ventricles commonly suffer alike. Other authorities say that the left is most often affected, then the right ventricle, right auricle and left auricle in rotation. Loomis states

that the auricles are affected alike. Hasenfeld and Von Feny-
vessy find, experimentally, that fatty degeneration does not
disable the heart muscle which responds as well as the normal
heart. In degeneration of the heart by phosphorus poison-
ing loss of compensation is manifest only shortly before death.
The failure of the heart is probably largely due to vaso-motor
weakness. The diagnosis must therefore be difficult, and the
fatty degeneration may be the result rather than the cause of
the failure.

The heart is usually enlarged, though this is apt to be
due principally to some associated lesion. When diffuse fatty
degeneration occurs alone there is frequently a moderate sim-
ple dilatation of the heart.

In association with, and allied to, fatty degeneration we
may have *brown atrophy* of the heart, in which there is atrophy
of the muscle fibres with the formation of granules of pigment.
It occurs in general marasmus, in senile conditions and occa-
sionally with hypertrophy. It has no clinical significance.

Granular degeneration.—In granular degeneration the
cells become swollen and their protoplasm granular. The
nuclei and striation of the fibres are lost. The heart tissue
is opaque, pale and soft. These changes eventuate in fatty
degeneration. In the heart they occur in connection with other
changes which together result in considerable enlargement
of the organ. This may be mainly or entirely dilatation, as
in post-typhoid, diffuse, granular degeneration. Again, in
parenchymatous nephritis immense hypertrophic dilatation
may develop in connection with the mixed forms of granular,
fatty and interstitial changes which result from the auto-intox-
ication due to imperfect oxidation and elimination.

CLINICAL HISTORY.—The clinical history of the various
degenerations of the heart is mainly that of more or less sud-
den loss of heart power, associated with those symptoms of
disturbance of other organs which result from, and sustain
the same ætiological relation to, the primary cause, as the
heart lesion.

In *fatty infiltration* there is a history of gradual increasing
shortness of breath, and a sense of oppression in the chest.
Drowsiness is often a prominent symptom; the patient will go

to sleep at all times, while sitting in a chair, and wake up suddenly without having been aroused, not realizing that he has been momentarily asleep. Fatty infiltration is seldom found before thirty years of age, and most often after fifty. It occurs in persons who exhibit the results of excessive and improper diet.

Fatty degeneration occurs in persons who are poorly nourished. In association with anæmia it occurs more often in women than in men, and in early life. The so-called idiopathic form is a disease of advanced life; it occurs most often in men and is usually associated with disease of the coronary arteries. Fatty degeneration is a process so intimately associated with other cardiac lesions that the only form having a specific clinical history is that occurring with anæmia or the infectious diseases. Here the sudden development of heart failure in a profoundly anæmic state, or during or immediately after the height of an infectious process, is indicative of fatty degeneration.

Granular degeneration.—In diseases attended with high temperature and systemic poisoning, signs of commencing heart failure may indicate granular degeneration. This condition may be recovered from before passing into a state of fatty degeneration. In parenchymatous nephritis the subjective signs of cardiac incompetency occurring within a short time (one to four months) of the probable onset of the kidney lesion, belong to the clinical history of this form of degeneration.

The occurrence of sudden death from degeneration of the heart is, in my experience, comparatively infrequent. Quain states that in fatty heart it is the most frequent mode of death.

Symptoms and diagnosis.—The symptoms of cardiac degeneration will vary with the ætiological associations. The one cardinal symptom is loss of dynamic power with or without cardiac enlargement, which, by exclusion is found to be due to muscular degeneration.

Fatty infiltration has no characteristic symptoms. There will be gradually increasing dyspnœa, slight cough, poor peripheral circulation, disinclination for mental or physical work, imperfect digestion and elimination and occasionally dizziness or a tendency to syncope.

The pulse is feeble, very compressible and is usually regular *when the patient is at rest*, but becomes rapid and possibly irregular also, on slight exertion. Later when degeneration of the muscle fibres is added to the infiltration, the pulse becomes more irregular and faster.

There may be slight enlargement of the heart, passive congestion of the base of the lungs, slight œdema of the limbs and the usual signs of failure of the left ventricle. The heart becomes considerably enlarged; the apex beat is diffused and indistinct and is displaced relatively more to the left than downward.

Both heart sounds are weak, the second being relatively louder than the first. The physical signs are essentially those of moderate cardiac dilatation, and the differentiation from dilatation due to other causes is largely one of exclusion. The area of cardiac dullness, and the disturbance of action, is greater in dilatation from degeneration than in dilatation from other causes.

Cheyne-Stokes respiration may or may not be present. It has been considered characteristic of fatty degeneration of the heart, but may occur in the asystolic period of any cardiac lesion.

The following history is from a case of fatty infiltration:

Man aged 58. Height five feet, seven inches, weight 212 pounds. Gradual increasing dyspnœa for a year. Tendency to go to sleep while sitting in chair at his desk. Mental effort difficult, memory clear. Indigestion. Bowels constipated. Heart slightly enlarged, apex under sixth rib in mammillary line, impulse diffuse and weak. Sounds indistinct, second louder than first. Pulse, sitting, 86; standing, 94; after slight exertion, 120; varies in force, not in rhythm. Slight passive congestion at base of left lung.

Improved under treatment, went back to business.

One year later relapse from overwork. Extensive dilatation. Death in two months.

Autopsy showed dilated heart covered with extensive layer of fat. The right ventricular wall appeared entirely fatty, its muscular fibres showed fatty degeneration under the microscope.

Fatty degeneration is so directly associated with the period of failure of the heart muscle of nearly all cardiopathies, that it is difficult to individualize its symptoms.

In elderly people who have developed arterial degeneration, and in whom the coronary circulation and nutrition is gradually but surely diminishing, there will be general failure of nutrition as shown by the cold skin, which is yellow and dry, and the presence of the *arcus senilis*. The slow peripheral circulation, irritable temper, failing memory, rapid, irregular pulse, tendency to sudden dyspnœa with slight cough after exertion, or even without, indicate progressive degeneration of the muscle. The pulse is quick, and hard, if there be much arterial degeneration. It is irregular in force and rhythm. It may resemble the pulse of chronic diffuse myocarditis.

The heart is usually enlarged from combined hypertrophy and dilatation. The apex impulse is diffuse and may be in the sixth interspace. When the degeneration is advanced the heart's impulse is weak and the apex difficult to locate.

In diffuse degeneration occurring with anæmia or the acute diseases there is rapid development of failure of the circulation with its attendant subjective signs, accompanied by rapid dilatation of the heart, as illustrated by the following history:

Woman, aged 41; sick eight months with pernicious anæmia. Treated with iron, arsenic and bone marrow without result. Blood count showed the red blood cells to be about two million per cubic millimetre and the oligochromæmia has averaged proportionally about the same as the oligocythæmia.

During the first month of her hospital life the pulse rate was from 95 to 100 per minute and regular. The heart was not enlarged when she entered the hospital. She gradually grew worse, pulse slowly rose to 140 per minute. Heart gradually dilated till left line of dullness was one-half inch farther to the left. Gastric disturbance, marked insomnia, dropsy of legs, failure of kidney action. Death. Autopsy showed fatty degeneration of heart muscle, dilatation of left and right ventricles.

Granular degeneration, as an initial process ending in fatty degeneration in acute diseases, may be suspected in case symptoms of failure of the heart in such connections are recovered from. Its chief clinical interest lies in its occurrence in connection with parenchymatous nephritis.

Along with the subjective signs of cardiac failure there is progressive enlargement of the heart, which is due to combined hypertrophy and dilatation. A very considerable enlargement of the organ may be attained in from one to four months.

Dilatation resulting from dynamical failure secondary to hypertrophy could not develop in so short a time, hence we have coincident dilation and hypertrophy which are concomitant with the kidney lesion.

The patient will suffer from œdema, passive congestion of the lungs and liver, cough, great dyspnœa, gastric distress. dizziness and faintness. The pulse, weak, soft and compressible, is usually regular, but may be irregular, and is not in proportion to the laboring action of the heart.

The heart may be much enlarged; its action is tumultuous. The apex beat is displaced outward and downward, its impulse is diffused and heaving in character. We do not get the direct, forcible, apex impulse of hypertrophy, such as is obtained with other forms of nephritis, but rather the diffused motion and indistinct apex beat of the hyposystolic period of advanced cardiopathies. We have here a patient exhibiting this form of cardiac disease who has been under treatment in the clinic for two months:

Girl 16 years old. Five months ago had scarletina; while convalescing developed acute nephritis; apparently recovered but did not regain strength. Developed shortness of breath, cough and œdema of the legs. Treated without relief. Appeared in clinic two months ago. At that time she suffered severely from dyspnœa and cough, could not walk across the room without great difficulty. Her lips and fingers were cyanosed. She could not lie down. There was extensive dropsy of the legs with passive congestion of the lungs and liver. Her pulse rate was 100-115 per minute. The heart's

action was tumultuous and disturbing. There was a widely diffused area of cardiac motion. The apex was in the seventh interspace, one inch and a half outside of the mammillary line. The transverse cardiac dullness reached from one-half inch to the right of the right edge of the sternum, to one-half inch to the left of the left mammillary line, on the level of the fourth rib.

The first heart sound was muffled, the second sound intensified, particularly over the aortic valves. She was passing fifteen ounces of urine daily, which contained albumen and casts. She has improved so that now she can lie down comfortably, sleeps well, eats well, coughs but little, walks at a moderate pace without disturbance. The œdema has entirely disappeared, also the passive congestions. The heart has diminished one inch in its transverse measure, and though, as you see, there is still an exaggerated area of cardiac motion, she is not conscious of the heart's action when she is quiet.

TREATMENT.—The treatment of degeneration of the heart embodies those principles which are applicable to the hyposystolic period of all cardiopathies.

In fatty infiltration, if the integrity of the muscle is fair and degeneration has probably not set in, we should give special attention to diet and exercise.

The carbohydrates should be restricted and the patient kept largely on animal food. Specific directions should be given as to the amount, as well as kind, of food to be taken.

The meats should be taken regularly and the same amount of food at each meal; not over ten ounces of fluid allowed with the meal. Mineral waters should be given between meals, to promote elimination. Exercise must be carefully graded to suit the condition of the heart. Walking, light exercise, calisthenics of any kind which do not increase the blood pressure too suddenly, will answer. Thyroid extract has lately been recommended for fatty infiltration in connection with general obesity. It is probably of value, but its definite contra-indications are not yet fully understood.

In more advanced cases where the muscle fibres of the heart are weakened from insufficient nutrition and we have a

mixed infiltration and degeneration, we must keep the dynamic condition of the muscle in mind. Here exercise may do harm, for the increased blood pressure will favor dilatation. We have the same condition in the fatty degeneration of senility and the treatment is the same.

All active exercise must be avoided. This is governed by the pulse; if the pulse becomes rapid or irregular, or there is dyspnœa, then the exercise is harmful.

Passive movements and very light resistance movements may be of service when performed with care, but they are apt to do harm and are not of the same utility as in degeneration secondary to valvular lesions.

Nutritious diet is important. Iron, arsenic and strychnia are the tonics to employ. The latter is of special value; it should be given in one-twentieth grain doses thrice daily and continued indefinitely. A degenerated heart muscle will probably never overcome the necessity for strychnia.

Arsenic is of particular value in cardiac degeneration. Alcohol is not admissible in any form.

Digitalis must be used very carefully, if at all. In beginning asystolism with serious dropsy and passive congestions, the careful use of digitalis may be followed by good results. A good infusion from the English leaf is the best and safest preparation to use.

Acute degeneration occurring from pernicious anæmia, the infectious diseases or poisoning, is not susceptible of much treatment. Beyond absolute rest and the energetic use of strychnia, not much can be done.

In granular degeneration of the heart associated with nephritis, elimination and nutrition are important. The cardiac condition is due to toxæmia from kidney incompetence. If the daily percentage of urea eliminated is less than that converted from the food, then the cardiac degeneration will be progressive.

The nitrogenized foods must therefore be restricted, the liver and intestinal secretions stimulated, milk diet given, with fruits and non-starchy vegetables.

It is surprising how much better the functions of the heart are carried on in these cases when occasional stimulation

is given the liver and bowels. A good combination for this purpose is a pill composed of three grains of mass hydrarg, one-half grain of euonymin, one-quarter grain of extract of nux vomica, and one and one-half grains of the compound extract of colocynth.

Iron and strychnia are the best heart tonics for these cases. Arsenic is not well borne as a rule.

The best form of iron is the mistura ferri et ammonii acetatis. The tasteless tincture of iron is also well borne.

The kidney lesion will require the use of such remedies as the diuretic salts of potash, strontium lactate, fluid extract of juniper, or Trosseau's diuretic wine.

Bouchard objects to potash as being actively concerned in the production of uræmic intoxication. We acknowledge that between its beneficial effect in promoting diuresis and its direful effect if not eliminated, there may be a therapeutic conundrum.

When dropsy and passive congestion are the features of the case special stimulation of the kidneys will be necessary, with such remedies as diuretin or calomel.

It is useless, however, to give these remedies with a very low blood pressure. The heart must first be stimulated and the blood pushed through the kidneys before these remedies can act.

Digitalis should therefore be given for three or four days previous to using special stimulation to the kidneys. The infusion is the best preparation to use. Tablespoonful doses may be given every four to six hours. To each dose may be added ten grains of acetate of potash, and two to five drops of deodorized tincture of opium as a vaso-dilator.

Trosseau's wine is a very efficient combination for occasional use when the urine falls below twenty-six or twenty-eight ounces per diem, and moderate combined stimulation of the heart and kidney is required.

Diuretin and calomel cannot be depended on in these cases as they can in the dropsies due to the passive congestion of the kidneys incident to valvular lesions, but they are always of more or less service.

I recently saw a man in consultation who was extremely dropsical from parenchymatous nephritis and toxic degenera-

tion of the heart with marked hypertrophy and dilatation. After stimulation of the heart with digitalis the exhibition of diuretin and calomel produced such rapid elimination of water that the man went into a condition of collapse, with a pulse of one hundred and sixty, and was only revived by the introduction into his circulation of sufficient normal salt solution to restore the blood pressure.

Diuretin should be given in fifteen grain doses every four hours until eight doses have been given, and then stopped.

Calomel should be given in three grain doses three times a day for two days only, the bowels being kept quiet by five drops of deodorized tincture of opium exhibited half an hour after each dose of calomel.

When much degeneration of the heart has occurred we cannot hope for more than temporary relief, particularly owing to the fact that the cause—the kidney lesion, is progressive.

CHAPTER III.

ACUTE ENDOCARDITIS.

Inflammation of the endocardium may be acute or chronic. The ætiology of these forms is largely identical, while clinically and pathologically they present marked individual features. In most cases of chronic endocarditis the tissue changes are but the advanced stages of an acute process, yet there may never have been any acute symptoms manifested.

Acute endocarditis may be infectious or non-infectious in nature. See has claimed that endocarditis is always of bacterial origin. Many varieties of organisms have been demonstrated in the products of endocardial inflammation. Still, in many cases infection cannot be demonstrated. Endocarditis occurring during intra-uterine life, is found almost altogether in the right side of the heart (Orth claims that, as a primary affection, it is equally common on both sides in fœtal life), and is a frequent cause of congenital lesions of the orifices of the right heart, defects of the auricular or ventricular septa or of cardiac malformations.

After birth endocarditis is rare in the right heart.

Acute, non-infectious endocarditis is sometimes called exudative endocarditis. Acute, infectious endocarditis is called ulcerative, or malignant (Osler).

ÆTIOLOGY.—Acute rheumatism is the most frequent cause of endocarditis. According to Bamberger twenty per cent. of the cases of acute rheumatism are complicated by endocarditis, while Bellevue Hospital Reports show that it occurred in over thirty-three per cent. of their cases. Nephritis is a frequent cause of endocarditis. The essential fevers, the exanthemata, diphtheria, syphilis and any disease in which there is an altered or toxic condition of the blood, may cause endocarditis. Wunderlich places measles next to

rheumatism as a cause of endocarditis. The disease may also be of traumatic origin (Bamberger).

Infectious endocarditis occurs secondary to scarletina, diphtheria, measles, pyæmia, puerperal fever, croupous pneumonia or to any septic process, wherever located. Gonorrhœa may cause acute infection of the endocardium, and the gonococcus has been repeatedly demonstrated in the inflamed endocardium of verrucose endocarditis secondary to gonorrhœa (His, Leydon, Dauber and Borst, Stengle and others).

Almost all varieties of infectious organisms have been found in infectious endocarditis. Austin has identified the influenza bacillus in three cases of recent endocarditis. A new organism—micrococcus zymogenes—has been discovered by McCallum and Hastings in a case of endocarditis. Some observers regard some lesion of the endocardium previous to the invasion of organisms as necessary to the development of infectious endocarditis. This is probably true in the milder degrees of infection, for we are beginning to learn that infectious endocarditis is of more frequent occurrrence and variable intensity than was formerly supposed. While a large percentage of the cases of infectious endocarditis already reported show lesions of the endocardium previous to the infection, such lesion is not a prerequisite to the development of the most malignant type of the disease.

Frantzel states that malignant endocarditis may be acute, subacute or chronic. The valves of the right heart, and especially the pulmonic valves, are not so frequently affected as in other forms of endocarditis, and that the ventricular portion of the endocardium is relatively more often affected than in other forms of endocarditis.

Because of the association of malignant endocarditis with puerperal fever it is particularly liable to occur in women. Infectious endocarditis may occur secondary to typhoid fever (Shattuck), or to la grippe (Huchard, Oulmont, Barbier).

The occurrence of endocarditis by the direct extension of inflammations of the lung, pleura, pericardium or myocardium is very doubtful.

MORBID ANATOMY.—Endocarditis is usually limited to the region of the valves and orifices of the heart. Occasion-

ally it may extend to the adjacent portions of the cardiac walls. The portions of the valves most often affected are those which are most exposed to friction, i. e., the convex surface of the aortic valves, and the auricular surface of the mitral valves.

At the aortic valves the inflammation commences along the band of tissue stretching from the corpus arantii to the attached border of the valve. At the mitral valve the inflammation begins on a line stretching across the surface of the valves at a short distance from the insertion of the chordæ tendineæ.

Vegetations attached to the valves may produce inflammation of the mural endocardium by friction against it, but infection from the valvular lesion is probably the most frequent cause.

In acute endocarditis the deeper layers of the endocardium become infiltrated with new cells, the inter-cellular substance is softened or destroyed. The new tissue pushes up the endocardial layer and forms granulations or vegetations (papillary endocarditis). These granulations may lose their endothelium and induce a thrombotic deposit of fibrinous coagula.

If the sub-endothelial cell infiltration is very rapid and intense, the new tissue may become necrosed with loss of substance (ulcerative endocarditis). These ulcerations may extend to and involve the cardiac substance. If the cell accumulation is sufficiently rapid, small accumulations of pus may form in the deeper layers of the endocardium (endocardial abscess).

The term mycosis endocardii has been applied to ulcerative endocarditis because of the various bacteria and micrococci which may be present.

Ulceration may cause destruction of the substance of a valve leaflet and result in its perforation. Laceration or aneurism of the weakened valves may result from pressure.

Embolic processes, infectious or otherwise, may result especially in infectious endocarditis. These emboli vary greatly in size and effect. Ischæmia and gangrene of the lungs may result from embolic infarction; hemiplegia and secondary softening of the brain if the emboli are large, if

small, softening with evidence of obstructed circulation. Infarctions and suppurations in the liver, spleen and kidneys may occur. Capillary embolism may occur in different organs at the same time. Capillary embolism in the skin causes numerous ecchymotic spots and may result in cellulitis.

Malignant endocarditis is especially liable to attack hearts which are the subject of chronic endocarditis.

Severe forms of endocarditis are likely to be accompanied by more or less myocarditis, the combined effect being to greatly weaken the heart muscle, embarass its action and increase the liability to dilatation.

When the inflammatory process is moderately intense the new tissue becomes partially organized and may undergo fatty or calcareous degeneration with resultant thickening, shrinking and rigidity of the valves, the cusps of which may be adherent to each other or to the walls of the heart.

Extensive vegetations and papillary growths may form from continued development of the new tissue.

CLINICAL HISTORY.—The clinical history of acute endocarditis will vary with the nature of its cause. There is, perhaps, no affection of the heart in which the subjective signs may be more indefinite than in the moderately severe forms of acute endocarditis, either infectious or non-infectious. If the heart muscle is not affected there may not be a single rational symptom which can be excluded from those due to the affection with which the endocarditis may be associated.

When endocarditis occurs in connection with rheumatism or nephritis we may have a rise of temperature of one or two degrees, accompanied by a sense of constriction about the chest and discomfort about the præcordium. The patient complains of dyspnœa, and palpitation which is not appreciable by palpation, and lies somewhat inclined toward the left side.

The pulse may or may not be increased in rapidity with the onset of the endocarditis; it usually is rapid in children, in whom there may be restlessness, anxiety or delirium. Pain is not usually complained of, though there may be ill-defined pain in the præcordium.

In severe cases, with considerable involvement of the heart muscle, there may be distressing dyspnœa, palpitation with a

pulse not at all in proportion to the action of the heart, and a temperature of over 103° F.

Again, in acute rheumatism we may, while making daily examinations of the heart, observe the physical signs of endocarditis develop, while at the same time there may not be present a single rational sign that would call our attention to the heart.

In malignant endocarditis the patient is more critically ill. There are erratic and frequent chills and an irregular temperature. The general condition may resemble closely that of typhoid fever or acute miliary tuberculosis.

The occurrence of infectious emboli is strongly suggestive of ulcerative endocarditis. Embolism occurs more frequently in infectious than in non-infectious endocarditis, and is most apt to affect the spleen. A typhoid state may be present in severe cases of malignant endocarditis which, with jaundice, chills and irregular fever, reaching possibly 105 or 106° F., may simulate the icteric form of malarial fever.

There may be suppression of urine with delirium and coma. This type of disease may be associated with pneumonia.

With rupture of a valve there may occur sudden dyspnœa and cyanosis, with symptoms of multiple embolism.

In some instances the acute form of malignant endocarditis may pass into a sub-acute or chronic form (Fraentzel). Here there will be more or less general improvement with an irregular, slight elevation of temperature, frequent irregular chills which occur at continually increasing intervals, thus prolonging the course of the disease for months. Litten has described a form of endocarditis—non-septic, malignant form of endocarditis rheumatica—which may begin during an attack of acute rheumatism or of chorea, or may commence with rigors or a hæmorrhagic rash, and may last for weeks or months. The subjective symptoms are, at first, those of simple rheumatic endocarditis. Its course is similar to that of septic endocarditis. The irregularities of the chills, and the rapid heart action are characteristic.

We have here a patient exhibiting a common history for an ordinary acute, non-infectious endocarditis:

Young woman, sixteen years old, came to clinic one week ago complaining of rheumatism in the feet and hands. Never had rheumatism before this attack. Ankles swollen and tender; walks with difficulty. Second and third metacarpal joints in each hand swollen and tender. Temperature 100° F. Tongue coated, appetite poor, bowels regular. Pulse 110, irregular in force, regular in rhythm, irritable and quick in character.

On examination of her heart we find a soft, blowing, systolic murmur in the præcordial area, most intense just above the apex. No alterations in the heart.

Were this patient confined to bed the pulse would not be so fast and there would be nothing to direct attention to the heart, the condition of which would remain undiscovered unless through a practice of routine examinations.

Given a patient the subject of chronic endocarditis in whom there is a history of recent and more or less sudden disturbance of cardiac action, we must bear in mind the probability of such disturbance being due to a fresh attack of endocarditis, particularly if there is no history of over-exertion.

We must also bear in mind the possibility of such an attack being infectious in nature, especially if the patient be suffering from pneumonia, typhoid fever, influenza or any infectious process at the time of occurrence of the cardiac manifestations.

SYMPTOMS AND DIAGNOSIS.—The only positive symptoms of endocarditis are those obtained by physical examination, and from these the diagnosis must be made.

Where an acute endocarditis has developed we notice that the impulse of the heart against the chest wall is increased in force. The position of the apex impulse may be distinctly visible, particularly is this the case in children in whom there is apt to be undue cardiac excitement.

The total area of impulse may be increased either from forcible action of the heart due to excitement of the organ, from enlargements due to previous disease of the heart, or, in severe cases, to dilatation due to myocarditis.

The impulse may be regular or irregular, and if there is much cardiac enlargement, tumultuous. Later the apex beat

and cardiac impulse becomes more indistinct, but never as suddenly as in pericarditis.

Palpation at the onset of the disease determines the abrupt, forcible apex beat, and the variations in force which occur; the regularity or irregularity of the heart's action; and the tumultuous action which characterizes the later stages of severe cases when myocarditis has given rise to dilatation. At this time the quick, jerky pulse of the earlier stages has changed to a soft, small pulse in marked contrast to the laboring heart. An endocardial thrill may possibly be felt at times.

On percussion the area of cardiac dullness may be normal or increased. Increase of dullness may be due to dilatation from myocarditis or from extensive deposit of fibrinous masses in the heart, or to combined hypertrophy and dilatation from previous disease. Increase in the breadth of dullness occurring after a few days may be due to dilatation of the right heart from increase in the pressure in the pulmonary artery from insufficient discharge of the left auricle (Skoda).

On auscultation we find the heart sounds, in the beginning of the disease, louder, stronger and abrupt. The second aortic sound will be intensified. Later they may be weaker and indistinct and the second pulmonic sound may be intensified.

There may, or may not, be a murmur. Usually a murmur is the first positive sign of endocarditis. A murmur may be valvular or ventricular. It is produced by the passage of blood over the roughened endocardium. Niemeyer says that a murmur may occur with healthy valves, merely from increased tension. A murmur from acute endocarditis will be systolic in time, soft and blowing in character, and will be heard with greatest intensity below the lower border of the fourth rib toward the apex. The point of maximum intensity may vary at different examinations.

With valvular localization a murmur will present, to some extent, the special characteristics of valvular murmurs.

The most common valvular localization of acute endocarditis is at the mitral orifice. It is said to occur in fifty per cent. of the cases of acute endocarditis. In children acute endocarditis almost always affects the mitral valves.

There may be a well-marked murmur or merely a rough-

ening or prolongation of the first sound. The murmur is limited to the region between the apex and the anterior axillary line. It is not, as a rule, heard behind. It may be heard over the epigastrium.

In most cases of acute mitral endocarditis the lesion presents the characteristics of mitral regurgitation. Mitral stenotic murmurs are seldom well defined during the acute stage of the disease. In children stenosis and regurgitation are frequently combined.

There may be accentuation of the second pulmonic sound, doubling of the first heart's sound, or reduplication of the second sound, in mitral endocarditis.

Transient pulmonic or tricuspid murmurs may accompany mitral endocarditis. According to Loomis aortic murmurs are present in mitral endocarditis in sixteen per cent. of the cases; tricuspid murmurs in fifty per cent. of acute mitral endocarditis, with forty per cent. of acute aortic murmurs, in twenty-five per cent. of mitro-aortic murmurs, and in twelve per cent. of all cases of acute rheumatic endocarditis.

Aortic localization in acute rheumatic endocarditis is infrequent in early life. In adult life it is usually associated with vascular and nephritic lesions, and its acute stages are never well marked. In malignant endocarditis at any age the aortic valves are frequently affected, and combined lesions of the aortic and mitral valves are not uncommon.

In acute aortic endocarditis the second sound may be wanting over the carotids. At the very onset the second sound may be louder than normal, but soon becomes less so and may be replaced by a murmur indicative of regurgitation, which is frequently the first evidence we have of aortic localization. A systolic, aortic murmur in acute endocarditis is usually softer than in chronic endocarditis, though it may be musical at any stage.

Acute endocarditis is to be differentiated from pericarditis, inflammation of the aorta, and the functional murmurs of anæmia and fevers, while the infectious type of endocarditis may be confounded with typhoid fever, acute miliary tuberculosis and pyæmia or malarial infections.

In pericarditis there is apt to be more præcordial pain.

The pericardial friction sound is superficial in character, not distant; it is harsh in quality, grazing or rubbing, not soft and blowing; it is a to-and-fro sound, not necessarily rhythmical with the heart sounds; it is confined to the præcordial space, not having any direction of transmission; its intensity is more likely to vary with change of position; it will disappear with the occurrrence of an increased area of dullness (effusion).

Aortitis is apt to be associated with general arterial disease; there is pain at the base of the heart or through to the spine; the pulse is more rapid and the respirations apt to be quicker than in endocarditis. The first aortic sound is indistinct or lost, and if calcification has occurred the second sound will have an unusually loud, metallic ring.

The functional murmurs of fevers are heard at the base of the heart instead of the apex. They are much softer in quality than the basic murmurs of aortic localization in acute endocarditis.

The hæmic murmurs of anæmia are heard better above the level of the fourth rib than below. They have no direction of transmission, are very soft in quality and are prone to be inconstant. They are heard best along the left parasternal line from the third to the fifth costal cartilage and there may be associated murmurs in any of the larger arteries.

When an attack of acute endocarditis occurs in a heart which has already developed the secondary effects of chronic valvular lesions, the diagnosis may be very difficult. When, however, under such conditions, the fever persists after all active rheumatic manifestations have been allayed, with a rapid, excitable or irregular pulse. an acute inflammation of the endocardium may be looked for.

When malignant endocarditis fails to produce distinct cardiac symptoms, or engrafts itself upon an old endocarditis without particular modification of the symptoms of the chronic affair, it may be very difficult of diagnosis. The erratic chills and irregular temperature, with examination of the blood, will eliminate both typhoid and malarial fevers. Careful physical and bacteriological examination will differentiate it from tuberculosis.

The occurrence of sudden enlargement of the spleen,

albuminuria, hemiplegia, dyspnœa or cyanosis, is suggestive of infectious endocarditis during any septic process, especially if there has occurred acute insufficiency of the cardiac valves.

This patient exhibits an acute attack of endocarditis engrafted upon a chronic affair.

Man 36 years old. By occupation a laborer. Six years ago had acute rheumatism; recovered slowly; resumed work and experienced no trouble until three weeks ago. Was not aware of any cardiac difficulty subsequent to the first attack of rheumatism.

Three weeks ago he had a slight attack of rheumatism in the feet, which were swollen and painful. He suffered from dyspnœa and palpitation, and appeared at the clinic because of these troubles. Temperature 101° F. Pulse 110; very irregular in force, slightly so in rhythm. Heart enlarged; apex in sixth space one inch outside of the left mammillary line. There was an aortic diastolic murmur, also a mitral systolic murmur not transmitted beyond the anterior axillary line. The second pulmonic sound was accentuated.

The enlargement of the heart is due to hypertrophy and dilatation of the left ventricle from chronic aortic lesion.

The question which presented itself was, whether there was an acute attack of endocarditis in connection with chronic aortic endocarditis and its sequences, in which the former condition had caused weakness of the muscle and resulting dilatation; or if the hyposystolic period of a chronic valvular lesion had developed coincidently with an attack of acute rheumatism. In deciding this question the history was of little value, as the patient was not sure but that he had sustained heart strain from overwork just previous to the appearance of his symptoms.

He was placed on active anti-rheumatic treatment and at the end of ten days all symptoms of rheumatism had disappeared. His heart was still rapid and excitable with palpitation. The temperature was 99.5° F. He was sent to bed and placed on sodium iodide. His dyspnœa is not troublesome at present. Temperature normal, pulse eighty and regular. He does not suffer from palpitation unless he exerts himself.

You will see that while the aortic murmur is unchanged and there is still present the evidences of chronic enlargement of the heart, the mitral murmur is scarcely to be heard, which fact, together with the relief to the dyspnœa and palpitation, and a slight reduction in the width of cardiac dullness with a lessened force of the second pulmonic sound tends to show that the heart has partially recovered from the weakness caused by the attack of acute endocarditis.

TREATMENT.—The treatment of an attack of acute endocarditis will vary with the nature of its cause. The ætiological treatment embodies, if possible, more active treatment against the associated disease.

When acute endocarditis occurs with nephritis, elimination by the kidneys and bowels must be pushed. The skin cannot be used as a channel for elimination, as the results are not sufficient to warrant the necessary depression.

When acute endocarditis complicates acute rheumatism the latter must be brought under control as soon as possible.

It seems to be a common opinion that cardiac complication in acute rheumatism is less likely to occur when sodium salicylate is employed, than when salicylic acid is used.

I do not know of any way to decide such a question, but when the joint manifestations of acute rheumatism are very severe, I do not hesitate to take advantage of the quicker relief afforded by salicylic acid, even though chronic endocarditis be present, or the acute form should develop. The depression from the acid is at times marked, and the heart should be watched closely. Ten grains may be given in capsule every hour or two hours, until pain and swelling are relieved and the temperature lowered, then the sodium salt should be substituted.

In milder cases of rheumatism, even though endocarditis should develop, the sodium salt is the better remedy and shou be pushed, for we need not expect improvement in the endocarditis until the active rheumatic manifestations have subsided.

It is claimed by those who use the method advocated by Fuller for the treatment of acute rheumatism, that when the alkaline treatment is adopted cardiac complications are much less likely to occur. Bartholow states that this method is adapted to the treatment of rheumatism in plethoric subjects and is not well borne by anæmic patients with rheumatism.

In regard to the treatment of the endocarditis itself we have to choose between the expectant method comprising rest, diet and the management of the rheumatism, and the more active treatment directed towards limiting the extent and intensity of the inflammation. Rest is absolutely necessary no matter what method of treatment is adopted. A patient with acute endocarditis should be kept in bed for from three to five weeks and all exercise should be given by gentle massage. In early mitral stenosis Sansom advises massage exercises, short warm baths followed by cold sponging. Too much rest may enfeeble the heart in this particular trouble.

The diet should be easy of assimilation and intestinal fermentation should be avoided.

The internal antiphlogistic treatment of endocarditis has generally been discarded. There is no evidence that the internal administration of calomel will in any way modify the extent or severity of the inflammation.

Digitalis should not be used; its employment for tachycardia, or palpitation due to endocarditis, as has been recommended, is irrational. There is no condition in endocarditis, *per se,* which justifies the use of digitalis.

Sodium iodide (five to seven grains), or potassium iodide (eight to ten grains), should be given thrice daily during the course of the disease. If there is restlessness, from pain in the joints or from præcordial pain, we are justified in giving anodynes for its relief. Some form of opiate is most suitable for this purpose. Two or three leeches to the præcordial region may be used for pain if deemed advisable.

External applications to the præcordium are variously regarded. Sinapisms, vesicants, cups and leeches have generally fallen into disuse, though there is no doubt that each may have its value in certain instances.

Even when pain is present heat does not seem to afford the same relief which attends its use in the more superficial inflammation of pleurisy. Cold is more effective. The use of the ice bag is highly endorsed by Forchheimer, and I have seen much relief to the pain and palpitation from its use. In some cases in children, however, the application of cold increases the palpitation and distress.

Vesicants over the præcordium, as usually employed, do little good, and in children they are objectionable.

Caton strongly advises a form of continuous counter-irritation by means of a series of small blisters of the size of a florin, applied one at a time to the front and sides of the chest along the course of the third, fourth, fifth, or sixth intercostal nerves. Caton reports forty cases treated in this way, combined with prolonged rest and the internal administration of the iodides, with twenty-nine recoveries with normal hearts. This is a somewhat remarkable percentage of recoveries, and granting the complete recovery of fifteen per cent. of the cases under any form of treatment, is still a strong endorsement of the method. Caton's treatment is to keep the patient in bed, wrapped in flannel from the neck to the feet, and on a light diet, absolutely excluding nitrogenized food except milk. Internally he gives salicylates and some cholagogue.

In the recurrent form of acute endocarditis the therapy is directed against the systemic cause. Some gouty, rheumatic, nephritic or intestinal toxæmia will be found as the cause of this form of the disease, which needs much study and observation before special therapy can be instituted.

The treatment of infectious endocarditis is not very satisfactory. Sources of infection must be removed if possible.

Complications of hæmorrhages or infarctions must be managed according to location and extent.

On account of the disastrous effect of infectious endocarditis upon the myocardium the chief indications are stimulation and nourishment.

Alcohol, ether, camphor, musk, strychnia and quinine may be used according to indications. When heart failure threatens, the special stimulant selected should be administered hypodermically. Strychnia and alcohol are the best for this purpose. Nitro-glycerine should not be used unless there is high arterial pressure, and then it should be used cautiously.

Fraentzel recommends quinine and alcohol in ample and continuous doses in malignant endocarditis. He advises fror seven and one-half to fifteen grain doses of quinine two o' three times daily. It should not be given to the point of producing nervous disturbance.

On account of the possible effect of high temperature on the heart muscle, the administration of antipyretics to control the rise in temperature may be advisable in cases where the fever is severe. Here it is a choice between the bad effects of high temperature and the depressing effect of the remedy to be used. The latter is at times to be risked in preference to the former.

Antipyrin is too dangerous. Acetanilid and phenacetine are somewhat safer, especially the latter, when given in small doses (three grains) in connection with quinine.

Generally the coal tar products are objectionable in this connection. Small and frequent doses, however, especially when combined with a small quantity of quinine, produce less depression than large single doses. It is better to give three to five grains every three hours than to give eight to ten grains every six hours. The continued use of these remedies on consecutive days should be avoided if possible. If we can bridge over the active period of the disease and obtain time for the use of nourishment and tonics, we may possibly add to the list of recoveries of even the severe type of infectious endocarditis.

Before leaving this subject I wish to reiterate the importance of prolonged rest in the treatment of acute endocarditis. In the milder forms of acute non-infectious endocarditis the patients are particularly hard to control, and it is just these cases which develop insidiously into chronic endocarditis with valvular lesions which handicap the individual for the remainder of life, and place the physician in the position of a mere watcher to avert further evil. The impossibility of controlling the endocardial inflammation without absolute quiet, and the dangers of exertion, must be placed before the patient in a strong light, and the responsibility of disobedience made to rest with himself.

CHAPTER IV.

CHRONIC ENDOCARDITIS.

The majority of cases of cardiac disease which we are called upon to treat are the results, directly or indirectly, of chronic endocarditis.

The endocardial inflammation may not have been recognized until its sequences which we are called upon to trea disclose its existence. Through the influence of habit and of occupation, chronic endocarditis is more frequent in men than in women. It occurs at all periods of life and under all social conditions. Its insidious advent is responsible for delayed diagnoses. It is rather remarkable what extensive changes may occur in the heart as the result of chronic endocarditis unaccompanied by any history or subjective symptoms until the period of hyposystole or dynamical failure is reached.

ÆTIOLOGY.—The great majority of cases of chronic endocarditis are due to rheumatism and are the sequelæ of attacks of acute or subacute rheumatic endocarditis. Again, endocarditis may be a chronic process from the beginning, at least no acute period can be recognized. This form of the disease (contracting or sclerotic) is most likely to occur at the aortic orifice and be associated with the atheromatous or arterio-sclerotic degenerations of the aorta and systemic vessels incident to advancing age or connected with a gouty diathesis or with nephritic changes. (Jackson reports 100 autopsies in which sixty of the cases were due to arterio-sclerosis, and over one-half of these were above fifty years of age.) When this insidious form of endocarditis is of rheumatic origin it usually affects the mitral valves, especially when occurring in children. In adults, however, and even in children in rare instances, it may affect the aortic valves.

Chronic endocarditis may result in Bright's disease, or any of the infectious diseases in which marked toxicity of the

blood is present.   In most of these cases it is probable that the
is a more or less acute stage at the inception of the endocardial
inflammation which being accompanied by no subjective signs
is not recognized. Chronic endocarditis as a sequel of acute
rheumatic endocarditis usually affects the mitral valves in chil-
dren and the aortic valves in adults.

Chorea, with or without rheumatic associations, is fre-
quently followed or accompanied by chronic endocarditis.   The
latter when associated with chorea in early life is more likely
to affect the aortic valves than is a purely rheumatic endo-
carditis occurring at the same period.

Rupture of the attachment of a segment of a healthy valve
or of the chordæ tendineæ may result in the development of the
same chain of mechanical sequences as follow endocarditis of
rheumatic origin.

The increased force with which the cardiac valves close
under the high blood pressure incident to very laborious occu-
pations, may result in sclerotic or atheromatous changes about
the aortic valves (Fothergill, Rosenstein).

According to Peacock mitral disease may result from
severe exertion.   The effect of long continued and frequent
strain in producing functional disturbance of the heart, also
muscular changes and dynamical failure, has been shown by
Da Costa, Taylor and others.

Regurgitation at the orifices of the heart may result from
enlargement of the valvular rings independently of valvular
disease; dilatation of the aorta may in this way cause aortic
regurgitation, and dilatation of the ventricles may in like man-
ner cause mitral or tricuspid regurgitation.   These conditions
may be temporary or permanent.

MORBID ANATOMY.—In chronic endocarditis cell infiltra-
tion is much less rapid and abundant than in the acute form.
There is a marked tendency toward the development of a
fibrillated structure, with less softening and disintegration of
the intercellular substance.

The result of this process is a fibroid thickening of the
endocardium with induration and contraction of the valves,
with adhesion of their cusps from the bases towards the apices,
causing narrowing of the orifice.   Papillary growths and vege-

tations may form on the valves and may undergo calcareous or partial fatty degeneration. These growths are situated upon the ventricular surface of the aortic valves and upon the auricular surface of the mitral or tricuspid valves. The chordæ tendineæ may be thickened and shortened, and every conceivable form of distortion of the valves and orifices may result from these processes. The valves may become adherent to the cardiac wall, causing extensive regurgitation, or the onward current may be alone affected from the extensive growths on the valves.

The valves may become so adherent that there is simply a central slit in a diaphragm which stretches across the opening. Retractions and adhesions may cause the mitral valves to assume the shape of a perforated cone admitting but the tip of the little finger, when the tips of three fingers should normally be admitted. The aortic opening may be so altered as to only admit a small pencil, or from the adhesion of long vegetations to the aortic wall, sudden and extensive regurgitation may occur. Atheromatous masses and calcareous nodules may form, particularly about the aortic opening. Valvular aneurism, softening, ulceration and destruction of the valves may occur.

Interstitial endocarditis affecting the endocardium of the heart cavities, may cause insufficiency of the valves from shortening of the calumnæ carnæ and the chordæ tendineæ. The heart wall may become indurated and thinned, resulting in aneurism of the cardiac wall, which usually occurs at the apex of the left ventricle. Aneurisms at the base and in the interventricular septum may be connected with valvular aneurisms. Communication may be established between the ventricles.

Various degrees of myocarditis may be associated with interstitial endocarditis of the ventricular endocardium. Degeneration of the heart muscle develops both as the result of myocarditis and from the changes induced by the altered state of the coronary circulation, and acts through the latter cause as the direct means of producing rupture of compensation. In the latter case, perivascular sclerosis beginning around the branches of the coronary arteries in the case of aortic endocarditis, and around the venous branches of the coronary circula-

tion in the case of mitral endocarditis, interferes with nutrition and results in degeneration and dynamical failure of the muscle.

Fœtal endocarditis almost always occurs on the right side of the heart. It may be primary, but is usually secondary to congenital malformations of the heart. The latter, while of great interest, are apart from our present subject.

CLINICAL HISTORY.—The clinical history of chronic endocarditis up to the time when an ataxic condition of the heart is reached, is indefinite. The general effect of all valvular lesions, whether causing stenosis or insufficiency, is to cause stagnation of the blood current behind the lesion and a corresponding deficiency beyond the seat of the lesion. The result is increase in pressure in the venous system and lowering of pressure in the arterial system.

In order to overcome this condition of the circulation more pumping force must be furnished through the development of hypertrophy of the heart muscle, until *compensation* is established. The length of time that compensation will last depends on the constitution and habits of the individual, and will vary from a few months to a life time. During this period we may have no symptoms and we are often surprised to find valvular lesions which have evidently existed for years entirely unknown to the patient. It is remarkable what a large proportion of patients who exhibit marked endocardial disease deny absolutely ever having experienced any symptoms indicative of an acute endocarditis.

When compensation is not maintained, the circulatory disturbance in the various organs and tissues produce symptoms which are part of the clinical history of chronic endocarditis. Thrombosis in the heart cavities may occur from slowing of the circulation, and embolic infarction may occur in the pulmonary tract when the thrombus is in the right heart; or in the tract of the general arterial system, if thrombosis occurs in the left heart. Detached particles from a papillary endocarditis may cause like infarctions.

Sudden dyspnœa, cough, bloody expectoration with perhaps a chill (Rosenstein), may attend infarction of the lungs. Hemiplegia from cerebral embolism may occur in aortic endocarditis. Infarction of the spleen or kidneys may cause severe

pain in the region of these organs, but more often causes no symptoms. Hæmaturia with transient albuminuria may occur. Gangrene of one extremity or numbness with œdematous swelling may take place from obstructed circulation. Purpuric eruptions may occur from capillary embolism of the skin.

Dyspnœa is perhaps the most prominent feature of the earlier clinical history of chronic endocarditis. Appearing at first only on exertion, and varying much in degree it becomes gradually more severe and frequent. Congestion of the base of the lungs, particularly on the left side, from lack of power of the right ventricle, marks the approach of failure of compensation in mitral lesions and the asystolic period of aortic lesions. The weakening of the right ventricle is first shown by a dry cough with sibilant or sonorous rales, followed by fine, œdematous rales, with free expectoration. The expectoration may be bloody and quite free.

Orthopnœa now appears and may be quite troublesome. It may, indeed, appear early and before dyspnœa becomes particularly evident.

Pulmonary infarctions and apoplexy occur, in connection with which necrotic changes are more frequent than in benign embolic infarctions.

If the congestion is continued the pulmonary capillaries project into and encroach upon the alveolar spaces, the aërating surface is diminished and a more or less constant dyspnœa is established (cardiac asthma). The continued congestion of the pulmonary circulation causes increase of connective tissue, capillary hæmorrhage and pigment deposit (brown induration). Œdema of the lungs and bilateral hydrothorax may occur. All of these conditions contribute to the extent and permanency of the dyspnœa..

Dyspnœa will also occur from increased tension in the systemic arteries from contraction of the arterioles, in which case it is spasmodic and resembles a true asthmatic paroxysm. The patient, however, is unable to move about as every movement increases his distress.

Cyanosis may be present to a varying extent. The more marked degrees of cyanosis are likely to appear in aortic endocarditis when relative mitral regurgitation marks the development of the hyposystolic period.

Marked discomfort, with a sense of fullness in the epigastrium, may result from congestive enlargement of the liver, which may become large enough to fill two-thirds of the abdominal cavity. "Nutmeg" liver may result from long continued congestion. Jaundice may result from swelling of the liver or catarrh of the common bile duct. Congestion of the digestive tract produces marked disturbance of digestion, flatulence and alternating diarrhœa and constipation. Hæmorrhage may occur from piles, or hæmatemesis may occur. Menorrhagia or metrorrhagia may be present or there may be amenorrhœa if the quantity of blood is small. Epistaxis sometimes occurs and may be severe.

Scanty, high-colored urine of high specific gravity is a more or less constant feature of the history of these cases. The amount of solids in the urine will be near the normal if actual kidney disease is not present. Lithates will be deposited freely. A moderate amount of albumen will usually be found, its presence and quantity depending on the amount of congestion of the kidney. If the latter condition be marked, casts may be found in the urine, with possibly a few blood cells. Granular kidney may develop if the congestion of the kidney is sufficiently prolonged. In chronic endocarditis there is, in the absence of kidney lesion, a distinct relation between the integrity of circulation and the amount, specific gravity and color of the urine.

Œdema is another important feature of the history of all cardiopathies, and is one of great interest to the patient. It appears first toward night about the ankles and dorsum of the foot if the foot covering be loose. The tissues are at first puffy and then become doughy, pitting on firm pressure. The œdema gradually becomes more extensive and may involve the whole body, but is usually confined to the most dependent portions. It varies greatly in amount and constancy, depending on the condition of the circulation and the habits of the patient. Cardiac œdema has a soft, doughy feel and the skin unless hyperæmic is never as shiny as in nephritic œdema. In the latter, the tissue has a hard, indurated feel and the swelling may appear about the loins before it develops in the feet.

As to the cause of œdema, it is fair to presume that it

depends on increased exudation from the vessels and diminished absorption by the lymph channels, and that the extent of the œdema depends on the permeability of the vessel wall and the intra-capillary blood pressure (Starling). The nervous system probably has nothing to do with the production of œdema except in so far as it may influence capillary blood pressure.

Cardiac palpitation may be present in endocarditis, but is due to disturbed innervation or to pressure on the diaphragm from gastro-intestinal disturbance, rather than to the cardiac lesion. Palpitation is more indicative of functional than organic heart disease. Arrhythmia of the heart, if present, is generally due to reflex influence. It may be a feature of mitral stenosis. In advanced cardiac disease arrhythmia may be directly indicative of asystolism.

Pain is inconstant, and is an attribute of no valvular lesion, with the possible exception of some aortic lesions when they are associated with disease of the aorta and coronary vessels and the heart walls. According to Peter, pain in aortic disease is due to irritation of the basic cardiac plexus from dilatation of the first portion of the aorta.

Cheyne-Stokes respiration which occasionally presents itself in cardiac disease, belongs to the nervous phenomena and is usually a symptom of toxæmia, and indicative of the uræmic state. It appears late in the disease and is usually present in cases of endocarditis associated with nephritis. While of a serious prognostic import, Cheyne-Stokes respiration is not necessarily an evidence of fatality.

Fainting spells may be associated with the low arterial tension of aortic stenosis. Cerebral embolism and apoplexy occur with aortic lesions, and may be the cause of sudden death, though in my experience this is not frequent.

Before discussing the special valvular symptoms of chronic endocarditis, we may briefly consider *the relation of endocardial murmurs to chronic endocarditis.*

First as to the cause of a murmur. The "tension" theory, "friction" theory, or Corrigan's theory of the development of blood currents, are all insufficient. A more reliable theory is that adopted by Davidson, Geigel and others, that a murmur

results from the lateral vibration of the valve or the heart walls from the force of the impingement of the blood stream.

In differentiating endocardial from extra cardial murmurs, we must remember that the latter are seldom or never basic in situation, not necessarily synchronous with the cardiac sounds, usually obtain in the preventricular or parapexian region (Potain), have no fixed point of maximum intensity, no constant area of diffusion, are of median pitch and quality and are very changeable as to location, rhythm, pitch and constancy. Extracardiac murmurs of cardio-pulmonary origin are not necessarily suspended with interruption of respiration, as Laennec, their discoverer, supposed.

In distinguishing between organic and anorganic endocardial murmurs, we find that anorganic murmurs are almost always systolic, are heard best along the left parasternal line and toward the base of the heart, are not conveyed outside of the cardiac area, are softer (anæmic murmurs) in quality than organic murmurs and are likely to be more increased in intensity by assuming the recumbent position, than organic murmurs.

We must remember, however, that it is possible to have diastolic murmurs at either the aortic, mitral or tricuspid openings from stretching of the rings without organic lesion of the respective valves. In these cases we must depend on the secondary changes which have taken place in the heart to guide us.

A few cases have been reported of functional pre-systolic murmurs identical with the murmur of organic mitral stenosis.

Organic endocardial murmurs have a definite relation to the rhythm of the heart sounds, a more or less constant point of maximum intensity and direction of transmission, and have characteristic elements of pitch, quality and intensity. Sansom remarks on the possibility of mistaking an anorganic for an organic murmur when the sound is apical in situation and the patient is nervous and excitable. He emphasizes the necessity of careful auscultation in the sitting position after a period of rest and without removal of the clothing as tending to avert awkward mistakes (in insurance work, for example).

Sansom says that anorganic apical murmurs are heard with greatest intensity in the neighborhood of, rather than

immediately at, the apex; they are mesosystolic in time, soft in character and may exhibit a rhythmical crescendo and diminuendo in connection with respiration. He regards an
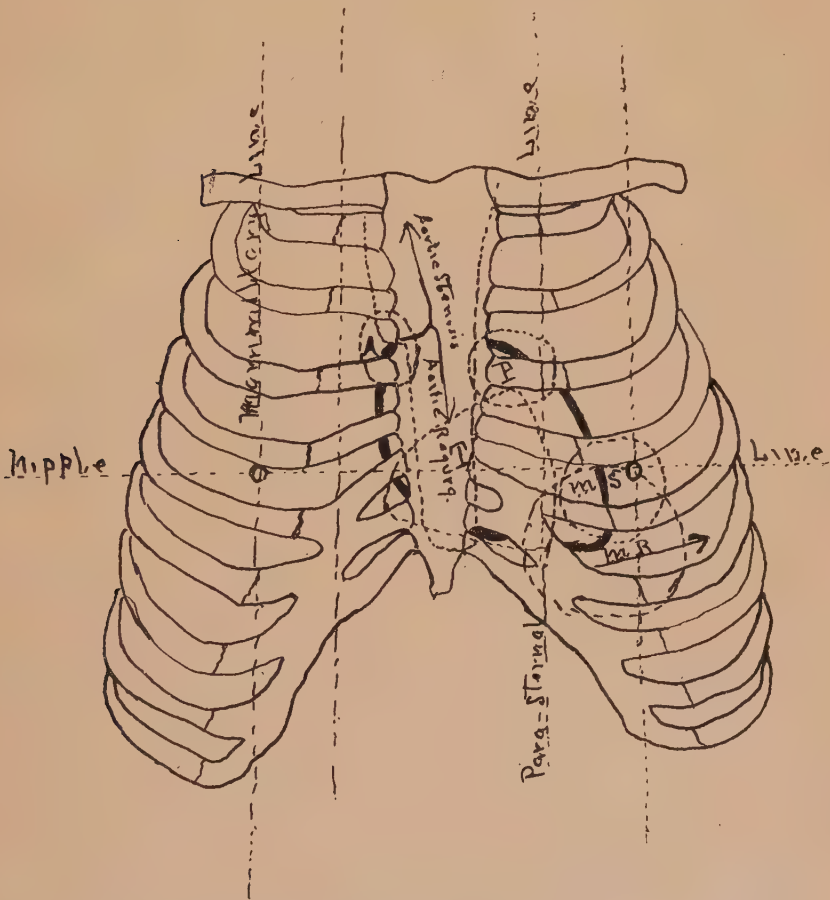
Outline of normal heart, and areas of auscultation for heart murmurs. A.—Aortic murmurs, arrows show direction of transmission. P.—Pulmonic murmurs. T.—Tricuspid murmurs. M. S.—Mitral stenotic murmurs. M. R.—Mitral regurgitant murmurs.

intensified second pulmonic sound, especially if connected with enlargement of the cardiac cavities, as decisive of the organic nature of a murmur.

It is difficult to judge of the extent of a valvular lesion from the nature of the murmur, and it is better to reserve our judgment until we see how the lesion is going to affect the heart. In the abstract, however, a long, loud, harsh, obstructive murmur, indicates considerable interference with the blood current; while a soft, weak, short, regurgitant murmur, indicates considerable reflux. Generally speaking, ill-defined murmurs are to be regarded with suspicion, and as indicative of either a badly damaged opening or a very weak heart muscle.

While the mutability of anorganic murmurs is much more pronounced than that of organic murmurs, we must remember that the latter may be changeable at times and may disappear completely for variable periods of time, without any demonstrable alteration in the physical character of the heart. The relative frequency of the disappearance of murmurs at the various valvular openings according to Musser is: first, mitral obstruction; second, aortic regurgitation; third, mitral regurgitation. No authoritative instances have been found of the disappearance of the murmur of aortic stenosis, or of tricuspid or pulmonary valvular disease. Again we may have chronic valvulitis with all the physical evidences of valvular disease except the murmur which may never be present during the history of the case. The absence of a murmur may render diagnosis very difficult. Fagge has applied the term *morbus cardii* to this class of cases and says they belong to the "sandy desert of cardiac pathology."

It becomes evident therefore that while a murmur is strong *prima facie* evidence of the existence of endocarditis, it is not *per se* absolute proof of its presence.

The diagnostic value of a murmur then, depends largely on the presence of such secondary changes in the heart itself, as may be looked for as the result of a specific valvular lesion. When a murmur is heard about a heart in which recent endocarditis can be excluded, and in which no secondary developments have occurred, the murmur is to be regarded as probably anorganic in character.

SYMPTOMS AND DIAGNOSIS.—In almost all instances the first symptom to call our attention to the presence of chronic endocarditis, will be a murmur. These murmurs have special

characteristics, and their respective lesions occasion definite changes in the heart. They will therefore be considered separately.

Aortic Stenosis. The mean age at which aortic stenosis occurs is said to be forty-seven, though it may occur in very young individuals. Men are affected more often than women, as in the former, from occupation and the greater frequency of arterial diseases, the aortic valves are under greater strain.

Aortic stenosis is usually combined with a greater or less degree of regurgitation, even though no regurgitant murmur be heard.

As the lesion of aortic stenosis causes interference with the escape of blood from the left ventricle, the increased intra-ventricular pressure during systole tends to cause dilatation which is opposed by hypertrophy of the left ventricular wall. This hypertrophy develops only to the extent necessary to overcome the obstruction at the aortic orifice, and the ventricular wall may become an inch thick. While there is, probably, always some increase in the size of the ventricular cavity, the condition is practically one of simple hypertrophy. Any marked degree of primary dilatation occurring in connection with aortic stenosis is (in the absence of myocarditis), due to concurrent aortic regurgitation.

In pure stenosis when compensation is once properly adjusted and conditions are favorable, failure of compensation may be deferred for many years.

In one case, a man with a well authenticated history of rheumatic endocarditis with aortic localization from a single attack of inflammatory rheumatism, compensation lasted thirty years. Autopsy confirmed the diagnosis of pure stenosis. The heart weighed nine hundred and twenty-five grammes. The man was six feet three inches tall and weighed two hundred and thirty pounds.

When muscular failure is developed, from causes already alluded to, the left ventricle is no longer able to stand the pressure, it dilates, relative mitral regurgitation occurs from stretching of the mitral ring and valves. The resistance thus

offered to the pulmonary circulation is not compensated for by the right heart because the latter is already too weak to comply with such a demand. Then follows right ventricular failure with the usual features of ataxia of the circulation.

The subjective symptoms of aortic stenosis previous to the period of involvement of the mitral valve, are few and indefinite. There may be palpitation or slight præcordial pain. The pulse rate is usually normal, the rhythm regular, though it may be intermittent or jerky in character. It is usually slow, hard and diminished in volume or fullness. It strikes the finger gradually and while it may be compressible is never soft. The sphygmograph shows a tracing with a slanting upstroke from weak percussion force, a blunted summit and ill defined secondary waves in descent, or wide separation of percussion and tidal waves.
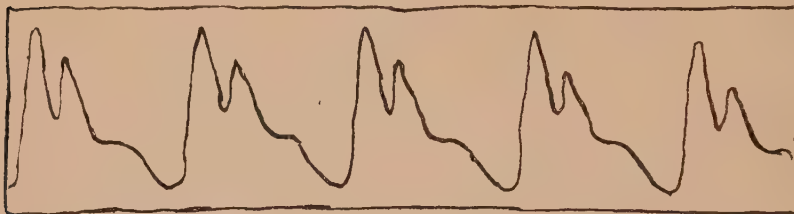
Tracing for a case of aortic stenosis, showing separation of percussion and tidal waves.

Faintness, syncope or dizziness from cerebral anæmia, especially on exertion, are the most characteristic subjective signs of aortic stenosis. These signs of arterial anæmia may be marked before the advent of rupture of compensation. When the latter occurs and the mitral valve becomes insufficient, the usual signs of venous engorgement appear.

Inspection will show—when compensation is perfect—an increased force and slightly increased area of apex beat, the position of which will be moved slightly to the left and downward. Later when dilatation occurs the area of cardiac motion may be greatly increased and may be heaving and irregular in character. Palpation determines the above features more distinctly. In the hyposystolic period the lateral displacement of the apex is greater than the vertical, as dilatation is now in

excess. A systolic *fremissement* may at times be felt in the second right intercostal space, it may radiate to the ensiform cartilage.

Percussion determines nothing unless dilatation of the left ventricle is present, as hypertrophy sufficient to compensate for a fair degree of stenosis will not enlarge the heart enough to give reliable evidence of the same on percussion. Later the relative enlargement of the various cavities may be made out.

On auscultation we hear the characteristic murmur of aortic stenosis : a systolic murmur heard with greatest intensity near the sternal border in the second right intercostal space or over the third costal cartilage. (If the heart is much enlarged, and displaced from its weight, the murmur may be best heard as low as the fourth rib and at the left border of the sternum.) The murmur is transmitted into the great vessels. It may be heard along the left subclavian artery and sometimes in the brachial artery and abdominal aorta. Behind it may be heard to the left of the spine about the third or fourth dorsal verte-bræ. In rare instances it may be heard all over the right chest. In front the murmur diminishes below the third rib, but may be heard as low as the xiphoid cartilage.

The murmur may be short or long. It is high-pitched and harsh in character and may be musical. I have heard an aortic stenotic murmur in a young girl that was audible at a distance of several feet from the patient.

The murmur of aortic stenosis is probably the most stable of all endocardial murmurs as regards location, character and constancy.

The following cases exemplify the lesion of aortic stenosis during the period of compensation, and also during the hypo-systolic period of the disease :

Man aged 41 years, carpenter by trade. Suffered from rheumatism eleven years ago. Never had but one attack of rheumatism. Suffers at times, when exerting himself, from dizziness or faintness which necessitates sitting down and rest-ing. At times has some palpitation of heart with a sense of disturbance in the præcordium. Never any distinct pain.

His pulse is 78, small and firm, and quite regular. There

are no passive congestions, no œdema, no cough, no dyspnœa.
The apex presents at the upper border of the sixth inter-
space one inch to the left of its usual situation. Percussion
determines that the left border of dullness is displaced about
an inch to the left. The right border remains unchanged.
The second pulmonic sound is not increased in intensity. A
loud, harsh, high-pitched murmur is heard with systole in the
aortic area. It is transmitted into the carotid artery and is
very faintly heard at the xiphoid region. Its character and
location are not modified by posture.

While there is a slight amount of dilatation of the left
ventricle in this case, probably due to slight regurgitation early
in the disease, the compensation is good at present and there
is no regurgitation to be recognized. The mitral valves are
competent and the case is *in statu quo*.

Girl aged 19, referred for treatment one year ago with
marked failure of circulation. Improved under treatment.
Failed to return until two weeks ago when her condition was
much worse than it had ever been.

The patient had several attacks of rheumatism when be-
tween 15 and 17 years of age. Has had slight rheumatic man-
ifestations ever since. Recently she has complained of dys-
pnœa, some cough, faintness and marked cardiac palpitation.
The palpitation has been extremely unpleasant to herself and
distresses her at night.

On inspection you notice the extensive area of cardiac
motion which is lifting and heaving in character. The apex
is in the sixth interspace slightly to the left of the mammillary
line. The left border of dullness is just outside the mammil-
lary line at the level of the fourth rib, and the right border
of dullness is one inch to the right of the right border of the
sternum.

A murmur is heard having the same characteristics as in
the former case, except it is not so harsh or well defined. The
second aortic sound is not clear, which indicates some regur-
gitation and which explains in part the dilatation of the left
ventricle. The second pulmonic sound is very loud and the

valves are evidently closing under great pressure. A long, soft, blowing, systolic murmur is heard in the mitral area, indicative of relative mitral regurgitation. There are a few œdematous rales to be heard at the base of the lungs.

This patient has developed rupture of compensation, and is passing from the hyposystolic period into a condition of asystolism. Through weakening of the heart muscle there has developed dilatation of the left ventricle, relative mitral regurgitation, dilatation of the right heart and asystolism is imminent. She will live but a short time. The arrhythmia is the irregularity of asystolism—a permanent inability of the heart cavities to empty themselves. One year ago this condition was threatening. There was a slight mitral leak with marked irregularity on exertion. Failure of the heart was averted by treatment, but her present condition is probably beyond the possibility of substantial improvement.

[This patient died two weeks afterward. There was slight improvement in the condition of the heart for a few days. Three days before death there was sudden pain, swelling, and great tenderness of the liver, probably from embolic infarction.]

Aortic Regurgitation.—Aortic regurgitation occurs without stenosis more frequently than the contrary condition. It is frequently associated with mitral lesions. According to Fagge only fifty per cent. of the cases of aortic regurgitation are of rheumatic origin. Virchow says that aortic regurgitation frequently occurs in chlorotic females from congenital causes.

The various conditions which cause atheroma of the aorta lead to aortic regurgitation. It may result from sudden and severe exertion. Dilation of the aorta may cause regurgitation either temporary or permanent. It is claimed that severe and prolonged muscular exertion may lead to aortic endocarditis resulting in regurgitation. Authentic cases are reported of distinct aortic regurgitation without organic lesions of the aortic valves.

As the lesion of aortic regurgitation allows of the return of blood to the left ventricle after its expulsion and during the

diastole of the ventricle, the latter cannot accommodate the additional amount of blood which is thus added to that nor-
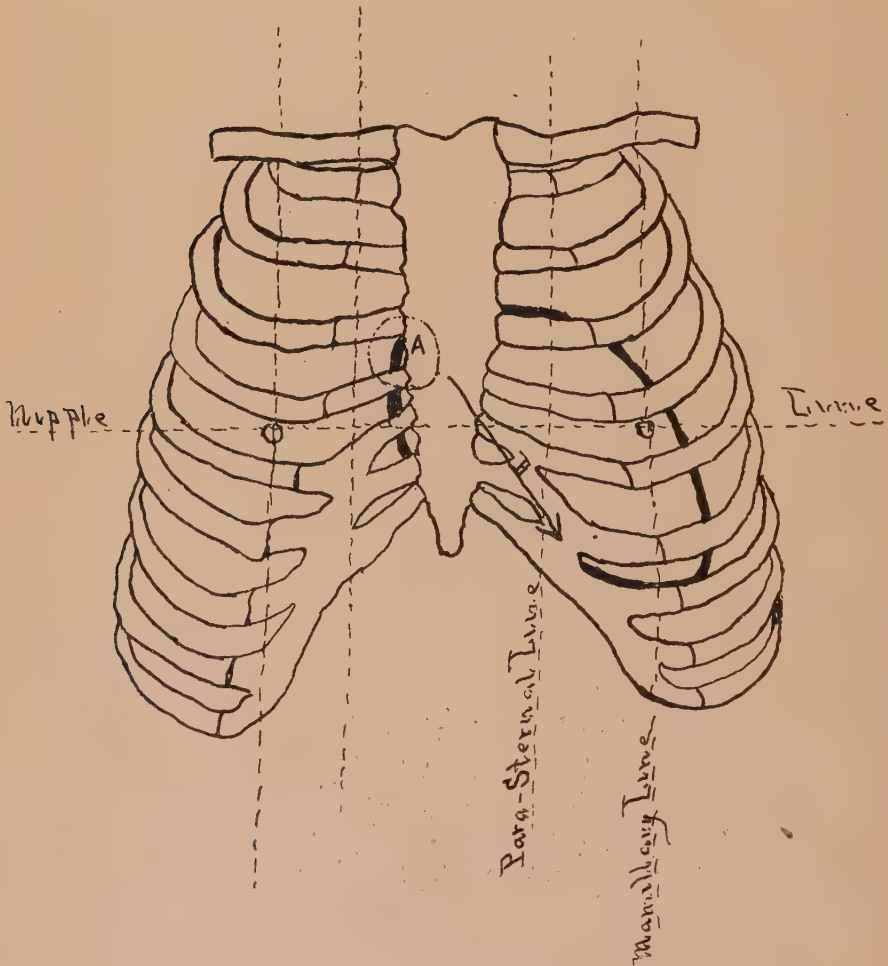
Enlargement of the heart in aortic regurgitation.　A.—Area of greatest intensity of murmur, somewhat depressed.　B.—Direction of transmission of murmur.

mally enterling the ventricle at the same time from the auricle. The ventricle therefore dilates, and to effect the expulsion of the greater amount of blood hypertrophy is necessary, and from

the combined development of these two conditions—dilatation and hypertrophy—we obtain an extensive degree of cardiac enlargement (the *cor bovinum* of the older writers).

While sufficient extra power is furnished through hypertrophy of the ventricular wall, this does not counteract the tendency to progressive dilation of the left ventricle if the leak is at all extensive. The dilatation gradually progresses until it becomes the dominant feature of the physical alteration in the heart. When the dilatation becomes sufficient there occurs mitral insufficiency, pulmonary congestion, dilatation of the right heart and the same attendant symptoms as are present in the asystolic period of aortic stenosis.

From a prognostic standpoint it is important to recognize the primary and progressive nature of the dilatation in aortic regurgitation. Even if compensatory hypertrophy is well established the ventricle is constantly overloaded during diastole and compensation cannot be so long maintained as in aortic stenosis where dilatation may be postponed until myocardial degeneration weakens the muscle.

The subjective symptoms of aortic regurgitation during the period of efficient compensation, are not distinctive. There may be palpitation with a sense of disturbed cardiac action and rapid respiration on exertion or excitement, or the patient may complain of dizziness, vertigo, headache or muscæ volitantes, and sleeps with his head elevated. There may be paroxysmal pain at the base of the heart (due according to Peter to irritation of the basic cardiac plexus from dilatation of the ascending portion of the aortic arch), which radiates to the left shoulder and arm. When dilatation is marked there may be disagreeable throbbing in the head. Attacks of angina pectoris may occur.

On inspection, we notice increased area of cardiac motion and the displacement of the apex beat to the left and downward. Relative to the amount of hypertrophy present the lateral displacement of the apex is much greater than in aortic stenosis. When dilatation becomes excessive the apex may be in the seventh interspace and at the anterior axillary line. The whole side of the chest may have a heaving, pulsating motion. When we see an apex beat in the seventh interspace and an

inch or two outside of the mammillary line we may be sure
at once that if the displacement be due to valvular lesion that
it is aortic regurgitation, for this position of the apex means
extensive hypertrophic dilatation of the left ventricle and no
other valvular lesion could produce this condition to the same
degree except aortic stenosis in its asystolic period.

Throbbing pulsation of the larger arteries, especially the
carotids, occurs when dilation of the ventricle is marked. The
great volume of blood thrown into the vessels distend them
suddenly and they as suddenly collapse and disappear. Capil-
lary pulsation of the cutaneous vessels may be seen by rubbing
the skin so as to produce a narrow line of hyperæmia. It is
also observed in the matrices beneath the nails (Quincke and
Ruault). When the arteries have become lengthened and tor-
tuous a lateral motion may be observed, especially in the
brachial, temporal or retinal vessels. Percussion shows that
the right border of dullness is little if at all changed while the
left is greatly displaced. The basic line of dullness may be
lowered an inch or more from gravitation of the enlarged heart.

Palpation determines the lifting nature and extent of the
cardiac motion. The area of apex impulse while extensive
is not well defined as in aortic stenosis. The apex does not
strike the finger as definitely as in the latter condition. A
systolic thrill may be felt over the subclavian or carotid arteries
due to the violence of the systolic impulse.

The pulse of aortic regurgitation has more individuality
than that of any other valvular lesion. It is a large pulse
and strikes the palpating finger suddenly. The wave is short
and drops away from the finger quickly. It has been called
the "Corrigan pulse," ",Bellingham pulse," "water-hammer,"
"jerking," splashing," "collapsing," "piston" pulse. Its char-
acteristics are intensified by elevation of the arm. It is usually
regular, though when dilatation is excessive the pulse becomes
irregular both in force and rhythm, and when mitral insuffi-
ciency develops the pulse becomes smaller, more feeble and
intermittent. By firm palpation of the wrist marked pulsation
may be felt in the radial, ulnar and interosseous arteries. The
sphygmogram of aortic regurgitation shows great amplitude,
a beak summit, and absence of the dicrotic wave.

On auscultation we have a diastolic murmur. It accom
panies or follows the second sound, which may or may not be
audible in the aortic area. The second pulmonic sound can
usually be heard with more or less clearness.

The murmur is heard best at the right edge of the sternum
at the upper border of the third rib, or, if the heart is much
enlarged, at the level of the fourth rib at the left sternal border.
It is sometimes inaudible at the base of the heart and heard
only below the fourth rib at the middle or left edge of the
sternum. Its direction of transmission is toward the end of
the sternum and the apex of the heart. It is rarely heard
behind. The arterial murmurs which are sometimes heard are
not due to transmission of this murmur as is stated by Loomis.
Like the double crural sound of Traube, and the double
murmur of Durosier, they are due to vibration in the arterial
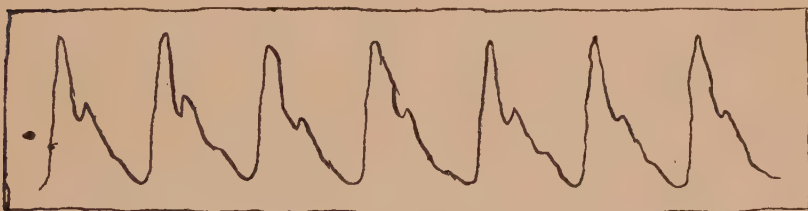
Tracing from a case of aortic regurgitation, showing absence of
dicrotic wave.

walls and depend on the force of the arterial distension, and on
lateral pressure on the vessels.

The first heart sound at the apex may be loud or indis-
tinct. According to Traube loss of the first sound is due to
gentle closing of the mitral valves under the increased intra-
ventricular pressure and before the systole actually begins. A
mitral murmur may occur from vibration of the valves without
relative regurgitation (Shattuck).

In character, the murmur of the aortic regurgitation is
soft, low in pitch, blowing or swishing, usually long though it
may be abrupt and short. It is very rarely high-pitched and
musical. The prediastolic murmur (Flint's murmur), said to
be associated with aortic regurgitation, is rarely to be heard.
It has never presented itself clearly in any of my cases.

Most authorities agree that the murmur of aortic regurgitation is the most constant and unchangeable of all murmurs. A number of observers, however, have reported cases of vanishing murmur of aortic regurgitation where autopsy proved the existence of aortic lesion.

The following case is of interest in this connection, though its nature is unproved as the patient is in good health, and we must remember, as Weismayr has lately pointed out, that the symptom-complex of aortic regurgitation may be due to accidental diastolic murmur in the aortic area; incompetency, without murmur; or to pseudo-insufficiency, without murmur or lesion :

Young woman, rheumatic history. Was told by her physician that she had heart trouble and cautioned against marriage. Five years later on contracting an engagement of marriage she came for advice. She presented a well-marked and characteristic murmur of aortic regurgitation, with a not very characteristic pulse. There was only slight enlargement of the left ventricle. The lesion was not deemed dangerous as there had been so little change during five years, and she was not discouraged from marriage which was shortly afterward contracted. Three years later the murmur had entirely disappeared, and during the last five years has not been present at a number of examinations. There has been, in the meantime, no change in the physical condition of the heart.

The following cases illustrate the difference in the progress of a slight and an extensive aortic regurgitation :

Man aged 43, laborer, six years ago had severe inflammatory rheumatism. Previous to this attack was perfectly healthy. Was told by his physician that the attack of rheumatism had affected his heart. Recovery slow, but was finally able to resume work and suffered no trouble until one year ago when he began to have pain about the heart, palpitation and dyspnœa on exertion.

On entering the clinic he was markedly dyspnœic, had some oedema of the feet, congestion of the lower portion of

the lungs, a troublesome cough. His heart was greatly en-
larged from hypertrophic dilatation of the left ventricle. The
apex was in the seventh interspace at the anterior axillary line.
The right ventricle was dilated. There was a long, soft,
diastolic murmur at the base, conducted to the apex. There
was also a systolic, apical murmur due to relative mitral regur-
gitation.

In this case compensation lasted only five years. How
much longer it would have lasted if the man had been engaged
in less laborious occupation, it is difficult to say.

Woman, aged 42. Wife of a professional man. Had ir-
regular attacks of rheumatism at 20 and 25 years of age. First
came under observation when she was 30. At that time she
had an aortic regurgitant murmur associated with a presystolic
apical murmur. How long these had existed it was impossible
to ascertain definitely. There was slight enlargement of the
left ventricle, the apex being under the sixth rib three-fourths
of an inch to the left. There was moderate dilatation of the
left auricle and a slight thrill could be felt. There was occa-
sional pain around the heart, and at times an irregular pulse.
A few days ago, about twelve years after the original
examination, there was found practically no change in the
heart; the apex was in the sixth interspace one inch to the left.
The heart's action was somewhat irregular in force, but not in
rhythm. The patient expressed herself as having been com-
paratively comfortable for several years except when her heart
would become disturbed from excitement or worry. The same
murmurs were to be heard as at the original examination.

Mitral Stenosis. Mitral stenosis is a lesion of early life,
though it occurs at all periods. It is more frequent in women
than in men. It is usually the result of rheumatic endocarditis,
though it is said to occur from the extension of aortic valvulitis.
It is usually combined with more or less mitral regurgitation,
frequently with aortic lesions, and is practically always asso-
ciated with tricuspid stenosis, whether the latter be congenital
or not.

. The lesion may be due to papillary growths; connective
tissue growth with contraction, rigidity and calcareous degen-
eration of the flaps; or adhesion of the flaps with a button-
hole or funnel-shaped opening. The effect depends on the
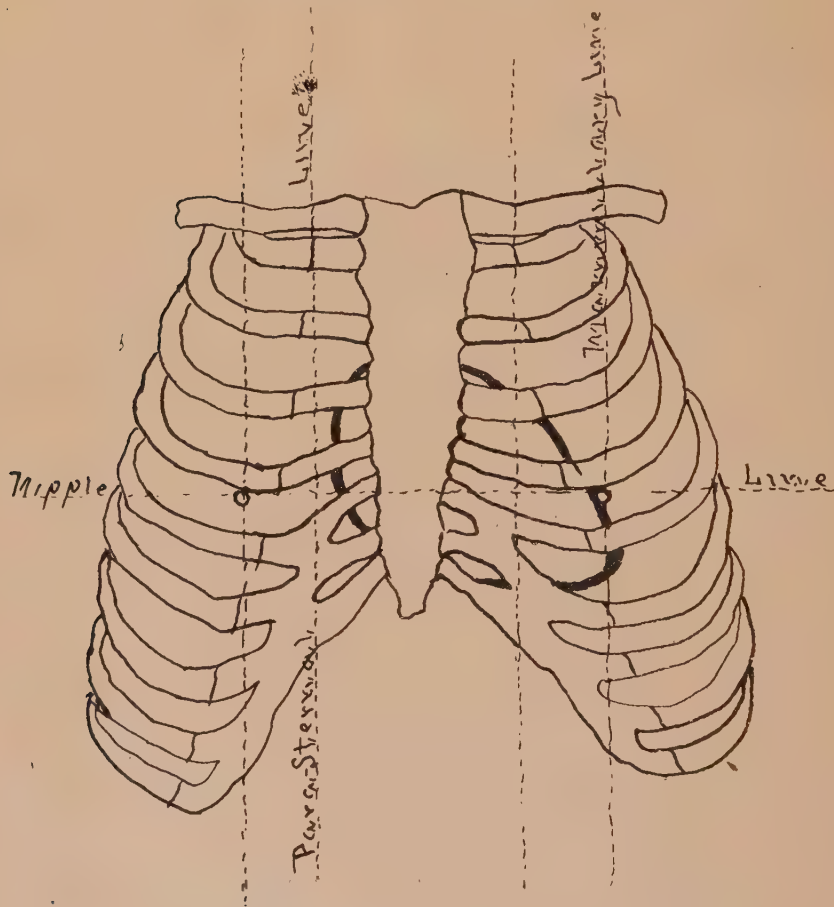
Enlargement of heart in mitral lesions.

degree of obstruction to the blood current. The resulting rise
in pressure in the left auricle and pulmonary vessels is unop-
posed and extends to the right ventricle, which furnishes the
compensating force. The left auricle may be considerably di-
lated, and its walls somewhat hypertrophied, usually not to any

great extent, though in some cases considerable hypertrophy may occur. The right ventricle becomes enlarged through hypertrophy and slight dilatation. Its walls may be as thick as those of the normal left ventricle, and do not collapse when incised. The right ventricle displaces the left in the formation of the apex of the heart. The left ventricle is not enlarged and may be smaller than normal owing to diminished intra-ventricular pressure. The aorta may also be smaller than usual for the same reason. In some cases the left ventricle may be enlarged, but this is due to associated conditions.

The pulmonary blood vessels may become thickened, and atheroma of the smaller branches may occur from the increased tension. The passive pulmonary hyperæmia may lead to brown induration of the lower portion of the lungs, or nodular infarctions, pulmonary apoplexy or œdema of the lungs may occur if the heart is suddenly overtaxed. When the right ventricular wall becomes weak and dilatation becomes exces-sive, tricuspid regurgitation will occur, followed by the usual effects of venous obstruction.

The resistance to the pulmonary circulation caused by mi-tral lesions, produces a serosity of the pulmonary tissues which is antagonistic to the development of tuberculosis, which rarely, if ever, develops in an individual the subject of a well-marked mitral lesion, and, when the latter develops coincident with or subsequent to tuberculosis of the lungs, the course of the latter is more favorable and the disease is more amenable to treat-ment. On the other hand, pulmonary tuberculosis frequently occurs in connection with pulmonic stenosis.

Rokitansky long ago remarked on the antagonism of mi-tral lesions and pulmonary tuberculosis as a principle governing the interrelation of these diseases. Von Weismmayr be-lieves this principle holds good and cites Frommold (21 cases in 226 of valvular disease), also Kryger (10 cases of valvular lesion in 1100 autopsies on tuberculosis patients). I have never seen pulmonary tuberculosis develop in a person the subject of well marked, chronic, mitral disease. Walsham found mitral stenosis only once in 21 post-mortem examina-tions of heart lesions associated with phthisis, and not at all in 1,000 cases of consumption observed clinically.

According to Allyn, mitral stenosis is the most unfavorable heart lesion in its relation to pregnancy. While opinions differ in this regard, it is probably a fact that the symptom complex of mitral stenosis is more unfavorably affected by the pregnant state than that of any other valvular lesion.

The subjective symptoms of mitral stenosis are not important. Women with mitral stenosis are usually of nervous, restless, irritable disposition. They are generally anæmic, and complain of gastric distress, which is most often due to the mechanical effects of the lesion on the circulation of the stomach.

Irregular pains about the region of the apex of the heart is a frequent cause of complaint. Palpitation and dyspnœa are troublesome on exertion. Walking against a strong wind will cause marked dyspnœa. Sudden and severe exertion may cause pulmonary œdema. Nervous influences of all kinds are extremely prolific in causing distressing palpitation. A dry, hacking cough simulating a nervous cough may be troublesome. Profuse, watery, mucous or blood-stained expectoration may indicate congestion or œdema of the lungs. Orthopnœa is not as troublesome as in other cardiac lesions.

Dropsy is an erratic symptom in mitral stenosis. According to Broadbent, cyanosis and dropsy in mitral stenosis indicate tricuspid regurgitation. While either of these symptoms is indicative of failing compensation, and dropsy may point towards the possibility of tricuspid regurgitation, it cannot be regarded as indicative of its existence.

Inspection shows some increase in the force and area of the apex beat with displacement to the left. The distinctly visible area of impulse has a sudden, jerky motion. The impulse of the left auricle may be visible in the third or fourth interspace inside the mammillary line. There will be epigastric pulsation if the right ventricle is much enlarged.

Percussion determines increase in the transverse dullness from extension of the right border of cardiac dullness which may reach the right nipple line. While compensation lasts this extension in the right border of dullness is usually not more than an inch. The apical dullness is rounded, displaced to the left, but remains at the same level. The left border of dullness

is displaced somewhat to the left and may extend upward and outward at the base from enlargement of the left auricle.

Palpation locates the apex beat and the abrupt and, at times, irregular character of the heart's action. A *thrill* may be felt in the region of the apex beat and is best determined by laying the finger lightly over the interspace. It is more the fine character of the thrill than the fact of its presence which is distinctive of stenosis. A thrill is not an invariable accompaniment of mitral stenosis, nor is it always indicative of the lesion. In some cases when there is considerable enlargement of the left auricle, the contractions of the auricle and ventricle may be separately appreciated by the fingers.

The pulse of mitral stenosis varies greatly in character. It may be regular and nearly normal in volume and tension. Usually it is more or less irregular, both in force and rhythm,

Pulse tracing in a case of mitral stenosis.

and is quick in character. According to Shattuck, and, indeed, most authorities, the pulse is regular except when disturbed innervation or compensation is present. In my experience the majority of cases of mitral stenosis exhibit a more or less persistent and characteristic arrhythmia which is independent of dynamical failure. How much disturbance of cardiac innervation may have to do with the arrhythmia is a question, though that it is a frequent cause is certain. Reflex nervous influence is more potent in the production of cardiac irregularity in mitral stenosis than in any other valvular lesion. Balfour states that irregularity of cardiac rhythm is always present to a greater or less degree in cases of mitral stenosis. The sphygmogram of mitral stenosis shows a rather pointed summit with a wavy line of descent. There is almost always more or less marked irregularity of the tracing. Inequality of the pulse in

corresponding arteries (*pulsus differens*) is often present in mitral stenosis. According to Popoff inequality of the pulse in mitral stenosis is due to pressure on the arteries by distended auricles and veins; competent tricuspid with distended left auricle and pulmonary veins affecting the left pulse; incompetent tricuspid with distention of venous trunks entering the right auricle, affecting the right pulse.

On auscultation we hear the characteristic murmur of mitral stenosis with an accentuated second pulmonic sound, which is sometimes reduplicated. True reduplication of the second sound is heard only at the base of the heart and is due to unequal pressure in the pulmonary and aortic systems—the valves do not close together. "Simulated" reduplication of the second sound is heard at the apex of the heart. The element of reduplication differs in time and character from true reduplication, and is probably produced at the mitral orifice or in the cavities of the left side, though the nature is uncertain. Balfour considers true reduplication of the second sound and a thrill as diagnostic in the absence of murmur. The second sound is usually plainly heard in the region of the apex. The first sound is abrupt and resembles the second sound. Later the second sound may disappear at the apex and the first sound become loud, short and sharp; following this the presystolic murmur may disappear and the short, sharp, first sound, with perhaps a tricuspid systolic murmur, will be all that can be heard. According to Broadbent, these signs correspond with the different stages in the dynamical condition of the heart in mitral stenosis.

The murmur of mitral stenosis is pre-systolic in time. In connection with the heart sounds it produces a triple rhythm. It is rough or vibratory in character. It may be longer than any other murmur when it occupies almost the entire period of diastole, or, it may be merely a short puff just preceding or accentuating the first sound. There may be a short interval between the murmur and the first sound, or the murmur may be continuous with the first sound. The murmur is best heard just above or inside of the apex beat and is synchronous with the thrill. Its area of diffusion is less than that of any other cardiac murmur, being confined, as a rule,

to the region of the apex.   In exceptional cases however, the murmur may be heard as low as the ninth rib or high in the axilla.

The murmur of mitral stenosis is probably the most variable of all valvular murmurs.   Walsh states that it may come and go from day to day, and all writers agree that it disappears with the development of dynamical failure.   Two conditions are necessary for the production of the murmur, i. e., roughness, or contraction of the opening and a sufficient amount of auricular contractility.   Dilatation of the mitral opening modifies the effect of the one cause, and variation in auricular pressure the other.   Variation in auricular pressure occurs frequently from causes which affect temporarily the contractile power of the auricle, hence the variable nature of this murmur.

The following history is from a case somewhat unique in the fact of an uncomplicated, pure, mitral stenosis in a man 62 years old:

Man aged 62.   Previous history unimportant.   Never had any distinct rheumatic manifestations.   During the last six or eight years has suffered more or less with dyspeptic troubles.   Is anæmic and thin, though never was rugged and strong.   For last two years has been troubled with cardiac palpitation and intermittent præcordial pains.   At times dyspnœa and some watery expectoration.   Lately has frequent dizzy spells or attacks of faintness.   Never had any dropsy.   Pulse rapid (195) and arrhythmic.

Heart apex in fifth interspace one-half inch to the left of normal situation, impulse slightly increased in force, quick and irregular in nature.   A distinct thrill is present a trifle to the right of the apex beat.

A loud, distinctly pre-systolic murmur is heard in the mitral area, which is not transmitted in any direction.   It is rough, vibratory and high in pitch.   There is a short but distinct interval between the murmur and the first sound.   The latter is rather short and more abrupt than usual.   The second pulmonic sound is intensified.

The character of the first sound, the second pulmonic sound, the thrill, the pre-systolic murmur and the arrhythmic heart's action in this case were quite characteristic.

Mitral Regurgitation.—This lesion is the most common of all valvular lesions. It is most frequent in early life but may occur at any age. It (mitral valvulitis) is usually primary though it may be secondary to aortic valvulitis. It is frequently combined with stenosis of the mitral orifice. *Relative* mitral regurgitation occurs as a mechanical consequence of dilatation of the left ventricle from stretching of the auriculo-ventricular opening. It is therefore a secondary effect of all conditions which cause dilatation of the left ventricle. The question is raised whether mitral regurgitation occurring from rheumatism is not due to muscle failure rather than to valvulitis. Fisher states that post mortem records show that pure and simple mitral regurgitation is a comparatively rare cause of death. Lees states that careful percussion will demonstrate general cardiac enlargement in acute and sub-acute rheumatism. Steele thinks that pure, mitral regurgitation of rheumatic origin results from muscle failure, which, with mitral stenosis is the usual result of rheumatism. Undoubtedly we have muscular changes in the heart associated with rheumatism and some cases of regurgitation may be due to this rather than to valvulitis, but that this is the rule cannot be accepted, especially in young subjects.

The mechanical effect on the heart of mitral regurgitation is similar to that of stenosis as far as the left auricle and right ventricle are concerned. The left auricle, however, does not become so greatly enlarged as in stenosis. The left ventricle becomes more or less dilated and hypertrophied. This is generally ascribed to the effect of the extra amount of blood entering the ventricle during diastole and the auricular contraction, this causes dilatation and there is subsequent compensatory hypertrophy. It is quite possible that in many instances the dilatation of the ventricle is largely due to muscular weakness from changes in the muscle caused by the rheumatism.

Compensation for mitral regurgitation is furnished through hypertrophy of the right ventricle. As long as the compensation is maintained there are few systemic effects; with its failure, however, we have the systemic evidence of failing circulation.

There are no subjective symptoms of mitral regurgitation

as long as compensation is well maintained, which may be for an indefinite period, neither patient nor physician suspecting the existence of a heart lesion. With loss of perfect compensation there will occur dyspnœa, a short, hacking cough, and frothy, watery or bloody expectoration. Exertion causes marked dyspnœa and cardiac palpitation. When backward pressure through the tricuspid valve occurs, there will be symptoms of venous stasis, cyanosis, enlargement of the liver with a sense of weight and fullness. There may be slight jaundice, nausea, anorexia, headache. Dizziness or stupor may occur from venous pressure. The urine is diminished in quantity, is high colored and deposits lithates freely. It may contain albumen and perhaps blood casts. Constant dyspnœa from pulmonary congestion and œdema with free watery or bloody expectoration will occur. The latter may be very plentiful if pulmonary congestion is great and frequently affords considerable relief to the patient. Œdema is a common symptom and is present in most advanced cases to greater or less extent. It may become very extensive and involve the entire body.

Inspection shows the area of cardiac impulse to be increased; it may be very extensive. In young children there may be prominence of the præcordium. A double cardiac impulse may rarely be seen (Bamberger, Skoda, Leyden). Epigastric impulse is visible if there is much enlargement of the right side of the heart.

Palpation determines the apex beat to be displaced more to the left than downward; it may be well outside the mammillary line and not lower than the sixth space. Again there may be little or no downward displacement while there is considerable lateral displacement. A systolic thrill or tremor may be felt at the apex, or perhaps at the base of the heart, but it is in no way distinctive of mitral regurgitation.

The pulse of mitral regurgitation is not strong in proportion to the heart's action, owing to the amount of regurgitated blood. It is soft, and regular as long as compensation lasts. When compensation begins to fail the pulse becomes irregular in force and rhythm and later becomes intermittent. These rhythmic disturbances of the pulse are often the first signs of failing compensation, and as this condition advances

they become very marked. The sphygmogram of mitral regurgitation is not characteristic. In advanced cases it will show simply an irregular, wavy line. When the heart's action is fair it shows amplitude, and breadth of the diastolic notch.

Percussion shows the extension of cardiac dullness to the right, as well as to the left and downwards.

On auscultation we obtain the characteristic murmur of mitral regurgitation; a systolic murmur which occupies a portion or the whole of the period of the first sound and more or less completely obscures it. The first sound—particularly in the early stage—may be clear and closely followed by the murmur. The murmur may be short but usually it is long. Its quality is soft and blowing, the pitch low, though exceptionally it may be high-pitched and rather harsh. Its area of diffusion is to the left. Usually the murmur is lost between the anterior
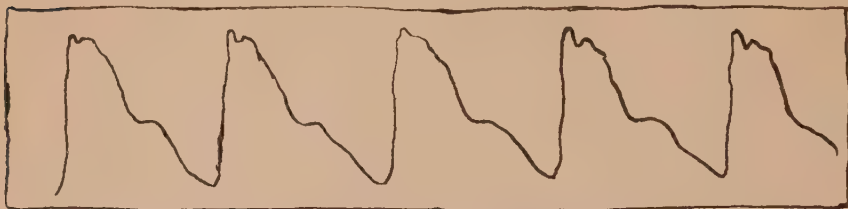
Pulse tracing in a case of mitral regurgitation in a well compensated state.

and mid-axillary line, to be heard again along the spine about the fifth and sixth dorsal vertebræ on the left side. In some cases the murmur is carried directly around the left side of the chest. Again it may be faintly diffused all over the front and rear of the left chest.

The murmur of organic mitral regurgitation varies much in intensity and character from time to time. It rarely disappears completely except with the advent of compensatory failure, or possibly when occurring in very young children. The so-called "dynamic" regurgitant murmur occurring in anæmia or chorea may disappear.

Relative mitral regurgitant murmurs are very soft, and, compared with their distinctness at the apex the extent of transmission is slight as they are seldom heard farther than four or five inches to the left of the apex.

During the period of compensation of a mitral regurgitation, the second pulmonic sound is accentuated. Skoda, who first emphasized this fact, considered it a "positive" sign of mitral regurgitation. It is certain evidence of high pulmonary pressure which is also present in mitral stenosis, though perhaps more variable in the latter condition.

The following history is from a case of simple uncomplicated mitral regurgitation:

Woman 42 years old has been under observation at different times for five years. Had rheumatism when 24 years old. No subsequent attacks of rheumatism. Married at 26, two children. Five years ago she presented herself in the following condition: Heart enlarged; right border one inch to the right of right border of sternum; left border at left mammillary line; apex in sixth interspace just outside mammillary line; blowing, systolic, mitral murmur; intensified second sound; passive congestion of the lower portion of both lungs; congestive enlargement of the liver the lower border of which was below the umbilicus; congestion of the stomach; œdema of both legs as far as the knees. She suffered from dyspnœa, cough and anorexia, and was expectorating freely a thin mucus. Orthopnœa was troublesome. Heart's action slightly irregular. She was relieved by treatment so as to feel quite well. During the subsequent five years she appeared for treatment four different times, each time presenting the same conditions as at her first appearance and each time being afforded the same relief. The last time she appeared the conditions were generally worse than before. Her subsequent history is unknown, but asystolism could not have been deferred for long.

Pulmonic Stenosis. This lesion is rare. The most experienced observers have seen but few cases. It is rarely seen in advanced life, being usually of congenital origin, in which connection it is usually associated with a patent foramen ovale. The majority of the reported cases of pulmonary stenosis have been in persons under thirty years of age.

The pulmonary valves may be fused together so as to have a very small opening. Tumors of the mediastinum, aneurisms or enlarged bronchial glands may press on the pulmonary

artery and produce in some degree the symptoms and effects of stenosis of the valves. The effect on the heart of pulmonary stenosis is to cause enlargement of the right ventricle from hypertrophy and dilatation. The tension in the pulmonary system is low and the lungs are frequently the seat of tuberculosis. Tricuspid insufficiency eventually results from dilatation of the right ventricle. In congenital cases the extent of the physical changes in the heart will be modified in proportion to the effect on the intraventricular pressure, of various degrees of patency of the foramen ovale or of the septum ventriculorum which may co-exist.

There are no rational symptoms specially indicative of pulmonary stenosis. Anæmia is frequently present; dyspnœa, cyanosis, cardiac palpitation and dropsy may be present.

Inspection and palpation will show some increase in the force and area of cardiac motion, slight displacement of the apex beat to the left, epigastric pulsation and perhaps irregularity of the heart's action. A systolic thrill may be felt in the second left intercostal space near the sternal border; according to Potain this is always present and easily recognized. Percussion determines extension of the cardiac dullness an inch or more to the right. Sight displacement of the left border of dullness may possibly be recognized. The basic dullness may reach to the second rib.

On auscultation we hear a systolic murmur. It is heard in the second, left intercostal space near the sternal border and is transmitted upward and outward toward the left shoulder. It is not heard toward the apex or in the vessels of the neck. According to Carpenter the murmur of mitral stenosis may be heard with greatest intensity anywhere between the second interspace and the level of the xiphoid cartilage. The murmur is usually plainly heard and is lower in pitch than stenotic murmurs of the left side of the heart. It is louder when the patient is lying down. Basic, systolic murmurs resembling that of pulmonic stenosis may occur from simple anæmia, dilatation of the pulmonary artery, communication between the aorta and pulmonary artery, and patent ductus arteriosus (Carpenter).

The following case presented the cardinal symptoms of

pulmonary stenosis in such association that an ante-mortem diagnosis was conjectural only:

Girl, aged 15. Not well developed, sickly since birth. Troubled with dyspnœa on slight exertion; cyanosis if exercises freely; no cough; more or less constant cardiac palpitation; slight clubbing of finger ends.

Examinations show moderate general enlargement of the heart. Basic dullness does not reach above third rib. Epigastric pulsation present. Heart's action irregular. Distinct systolic thrill in second, left intercostal space. Blowing, rather rough, systolic murmurs heard at the base and as low as the sixth costal cartilage over sternum. From second left interspace the murmur is transmitted outward and upward for a distance of three inches.

In this case autopsy subsequently proved the existence of congenital pulmonary stenosis, patent foramen ovale and deficient septum ventriculorum, a combination which would explain the symptoms and also the presence of a murmur as low as the sixth rib. The modification of intra-cardiac pressure through these communcations explains the comparatively small amount of cardiac enlargement. particularly as regards the right side.

Pulmonic Regurgitation. As an organic lesion pulmonary regurgitation is very rare and its occurrence is doubted by some. It may occur in connection with pulmonary stenosis. Relative insufficiency of the pulmonary valves is occasionally found. A number of instances are reported, most of which are supported by autopsy. Relative regurgitation may occur from disease of, pressure on, or plugging of (Litten) the pulmonary artery; diseases of the lungs or pleuræ (Gourand); or secondary to mitral disease (Gouget, Chaufford). Any cause which will raise the pressure in the pulmonary artery may induce pulmonary insufficiency. Myocardial disease will not cause regurgitation at the pulmonary valves as readily as it will at the other valvular openings, because the depth of the conus arteriosus modifies the effect on the valves of the dilatation which may result from muscular weakness.

There are no rational symptoms of pulmonary regurgitation, though some dyspnœa or cyanosis may be present. The physical signs will be those of enlargement of the right heart. There may be systolic pulsation in the second, left interspace, also a thrill, which may be systolic or diastolic. If the pulmonary artery is much dilated, the dullness at the base may extend to the second rib. The murmur of pulmonic regurgitation is diastolic in time, loudest at the second, left interspace and is transmitted down the sternum. According to Bernhardt it is loudest during expiration. Systolic intensification of the inspiratory murmur (Gerhardt's "audible capillary pulse") may be present but is not distinctive of pulmonic insufficiency. The murmur of pulmonary regurgitation may appear and disappear at times while under treatment (Pawinski).

I have in a few instances heard a diastolic murmur appear at the second left interspace in cases of valvular disease of the left heart, and in cases of pulmonary fibrosis. These murmurs were conveyed down the sternum and were followed by signs of dilatation of the left ventricle. As they could not be regarded as cases of tricuspid stenosis—especially in those instances where mitral disease was absent—they were probably cases of relative pulmonic insufficiency, though opportunity for a post mortem test of the valves was not had.

Tricuspid Stenosis. This lesion is very rare. According to Strumpell its occurrence is so rare as to be of no practical importance. Over one hundred and fifty cases are now on record supported by autopsy. It occurs more frequently in women than in men. About seventy-five per cent. of the recorded cases were in women, which Leudet thinks may be owing to the puerperal state. Tricuspid stenosis is usually associated with some other valvular lesion. According to Rosenstein, no case of tricuspid stenosis uncombined with other lesions is recorded. All of the cases collected by Fenwick show combined lesions. The most frequent association is mitral lesion. The next is mitral and aortic lesions (usually stenosis).

Uncombined tricuspid stenosis does occur, however, as is proved by Leudet's series, 114 cases of tricuspid stenosis, in eleven of which there was no associated lesion. About fifty

per cent. of the cases are due to rheumatism (Fenwick, Leudet), though Rosenstein and Peacock consider all cases as being of congenital origin.

Tricuspid stenosis may be congenital or acquired. The congenital form is usually associated with defects of the septum ventriculorum, patent foramen ovale and ductus Botalli, or pulmonary stenosis. In all cases of tricuspid stenosis there is usually some regurgitation. With marked stenosis the right ventricle may be small even though mitral stenosis be present, though as a rule it is enlarged, as is the heart generally, from associated lesions. The right auricle is usually enlarged, it may be greatly so, from hypertrophy and dilatation. While one or two cases of tricuspid stenosis have reached an age above fifty years, the majority die early. Over thirty per cent. die between twenty and thirty years of age.

The subjective symptoms of tricuspid stenosis are not distinctive. Cyanosis is generally present, as is dyspnœa, which may be severe. Cardiac palpitation and arrhythmia may be present. More or less marked evidence of venous stasis and œdema will occur. Presystolic venous pulsation is present, with at times a systolic pulsation from regurgitation. Systolic pulsation in the liver may be present.

Physical examination will show enlargement of the heart, which is usually general. There is nothing distinctive about the enlargement unless unusually high extension of the basic dullness on the right would indicate enlargement of the right auricle. The pulse of tricuspid stenosis is feeble and may be rapid and arrhythmic. Thrills may be felt at times. The murmur of tricuspid stenosis is diastolic. It is not constant or uniform in character, quality or intensity. According to Walshe, Flint and Hope, it is very rarely heard. Potain, Paul and others do not recognize its authenticity. Its point of maximum intensity is over the tricuspid area (beneath the sternum at the level of the fourth rib), and according to Grawitz is particularly diagnostic if heard at the right edge of the sternum over the fifth or sixth cartilages. It is a difficult matter to outline the features of such a murmur clearly, as associated murmurs, as well as arrhythmic conditions or tachycardia, will interfere with its recognition. In some cases the only murmur

recorded in the tricuspid area was a systolic one. Stokes, Duroziez, Fenwick, Duckworth and others say the murmur is diastolic. Hayden thinks that an open area between the characteristic situation of mitral and tricuspid diastolic murmurs, in which no murmur is heard, serves to distinguish and diagnosticate the two murmurs.

The diagnosis of tricuspid stenosis has seldom been made *intra vitam* (six times in one hundred and fourteen cases). The most characteristic sign is a diastolic murmur heard over the lower portion of the sternum, which can be differentiated from a mitral murmur. According to Shattuck, in a female patient with mitral or aortic disease and rheumatic history, with recurrent or prolonged venous stasis, tricuspid stenosis may be inferred whether a presystolic murmur is heard or not.

The following history is from a case of acquired tricuspid stenosis in connection with mitral stenosis:

Woman aged 24. Rheumatic history. Complains of some cough, dyspnœa, cardiac palpitation, præcordial pain, dizziness and faintness, and at times slight cyanosis. Examination shows general enlargement of the heart, especially of the right ventricle. The pulse is rapid and arrhythmic, feeble and of low tension. A mitral thrill in mitral area; a basic, systolic thrill is also felt. Slight presystolic, venous pulsation just above sternum. Presystolic murmur in mitral area. Over right edge of lower part of sternum can be head a diastolic murmur coincident with, but not quite as long as the mitral murmur. One-half inch to the left of the sternum, about the fifth interspace, there is an area where the murmur is very faint, but at no point between the mitral and tricuspid areas is it entirely absent. There is moderate congestion of the liver and stomach, also moderate œdema of the legs.

While tricuspid stenosis was suspected in this case it was impossible to separate the murmurs, and the general symptoms were not incompatible with the history of advanced mitral stenosis. Autopsy subsequently proved the existence of tricuspid and mitral stenosis. The total absence of any symptoms during the patient's early life previous to rheumatism, together

with the absence of any congenital defects or malformations of the heart, would indicate the acquired nature of the lesion.

Tricuspid Regurgitation. As a primary lesion tricuspid regurgitation is rare. Secondary to those conditions which raise the pressure in the right ventricle it is very common. These conditions are chiefly valvular disease of the left heart and particularly mitral lesions, and chronic diseases of the lungs and pleuræ (according to Balfour bronchitis ranks next to mitral lesion as a cause of tricuspid regurgitation). The occurrence of secondary tricuspid regurgitation marks the development of asystolism of the right ventricle. When it occurs from overtaxing the heart in connection with a primary mitral lesion it may be recovered from, but when mitral insufficiency secondary to the aortic lesion is followed by tricuspid regurgitation, the heart muscle is usually too weak to allow of any recuperation.

Valvulitis at the tricuspid opening is very rare in the adult. Ulcerative endocarditis is very rare at these valves. The morbid processes are not essentially different from those incident to valvulitis of the left heart. The effect of tricuspid regurgitation is to cause dilatation of the right auricle. In primary tricuspid valvulitis there may occur some hypertrophy of the right auricle, but in secondary regurgitation hypertrophy will not occur. The backward pressure of the regurgitant current is transmitted to the veins entering the right auricle. In the veins tributary to the inferior vena cava having no valves, the backward pressure is manifested. Enlargement of the liver may occur. In long continued cases "nutmeg" liver may develop. Enlargement of the spleen, congestion of the stomach, hæmorrhoids and induration of the kidneys will occur.

The subjective signs of tricuspid regurgitation are intermingled with those of the associated lesions in such a manner that it is difficult to separate them. There will be dyspnœa, orthopnœa, cardiac palpitation and arrhythmia, headache, vertigo and dizziness from passive cerebral hyperæmia. Gastric disturbances are sometimes severe. The skin becomes a dingy yellow. The kidney secretion is scanty and high-colored. Constipation may be obstinate and there will be dropsy with or without ascites. Broadbent thinks that œdema and cyanosis

in connection with mitral stenosis is indicative of tricuspid regurgitation.

Physical examination usually shows general enlargement of the heart which may be very great in some cases of secondary tricuspid regurgitation. Extension of dullness towards the right nipple and upward at the right base indicates enlargement of the right side. Systolic pulsation in the liver may be seen or felt. (This must be distinguished from the epigastric pulsation due to the impulse of the enlarged right ventricle.) Epigastric pulsation is usually present. Pulsation in the jugular veins is an important symptom. It may not be present at first unless the valves in the veins are situated an inch or so above the mouth of the vein, in which case pulsation may be seen just above the sternum while the valves are yet competent. Later with incompetent valves pulsation of the jugulars is marked. True jugular pulsation is distinguished from false or transmitted motion by pressing on the vein. If the pulsation is from the right auricle it continues between the finger and the heart and ceases above the finger.

The murmur of tricuspid regurgitation is systolic in time, low in pitch, blowing and soft in character, and is heard with greatest intensity at the left border of the sternum or over its lower part from the fourth to the sixth rib. It has a short direction of transmission upwards and to the right. It is seldom heard above the third rib and is not conveyed to the left. It is sometimes heard plainest over the right costo-xiphoid notch. This murmur together with epigastric pulsation and jugular pulsation constitutes the chief evidence of tricuspid regurgitation. While tricuspid regurgitation is of great clinical importance as an evidence of right ventricular asystolism, its symptoms are so mixed with those of the causative lesion that citation of cases would be but repetition.

PROGNOSIS. No definite statements can be made relative to the prognosis of a certain lesion without knowledge of the effect on the heart chambers or muscle which the particular lesion is having, this, of course, requires time. Mixed lesions are, of course, more unfavorable than single ones. Pure aortic stenosis and simple mitral regurgitation are undoubtedly the most favorable lesions. Between mitral stenosis and aortic

regurgitation it is not easy to choose; different observers vary much in opinion. However, discussion on this question is useless; the variations depend on the extent and effect of the lesion, and we can only prognosticate after understanding these. When a sufficient length of time has elapsed to show that there is little or no change taking place in the physical state of the heart as a result of the lesion, then if the dynamical condition of the heart muscle is good and the patient tractable and understands his limitations relative to physical effort, there is no reason why his life expectancy—other things being equal—should not be equal to that of any other individual.

CHAPTER V.

CHRONIC ENDOCARDITIS (Continued).

TREATMENT.—The treatment of chronic endocarditis embodies the management of the various degrees and phases of ataxia of the heart muscle into which the physical alterations of the heart which are secondary to valvular lesions eventuate. Neither the presence of chronic endocarditis, of a valvular murmur, or of physical alterations in the heart, warrant the institution of therapeutical measures unless as a result of one of these conditions there is dynamical failure of the heart muscle. A patient is not treated because he has a murmur, but because as a result of the lesion (and consequent upon the physical alterations in the heart consecutive to the lesion) there is inability of the heart as a pump. Until there is evidence of this failure of the heart muscle to cope with the demand upon it, treatment is unnecessary.

The length of time between the establishment of a chronic endocarditis and the beginning of muscular ataxia resulting therefrom, will, of course, vary greatly with the different lesions and in different individuals. I have known this period to last thirty years in an uncomplicated case of aortic stenosis.

The management of this period of compensation rests with the patient, who, if intelligent, may do much to avert subsequent trouble, or at least to postpone it if the nature of the trouble be explained and he be made to understand the limitations which apply to his individual case. A person with well compensated mitral regurgitation or aortic stenosis (the most favorable lesions) may follow any of the ordinary avocations, including those of the laboring man, without danger, providing he be careful not to undertake anything necessitating sudden, severe or continued strain. Persons with aortic regurgitation should not undertake hard physical labor, though light outdoor employment is favorable. Indoor occupation is, as a

rule, best adapted to cases of mitral stenosis, especially in changeable climates. These patients should avoid everything which increases the respiratory rate, also all mental overwork, worry or excitement, as these influences disturb the cardiac rhythm in mitral stenosis, more than in any other valvular lesion. The kinds of employment to be interdicted are those which entail exposure to cold and wet, severe muscular exertion, the breathing of impure air, the occupation of mining (because of tendency to develop anæmia), also that of sailor or soldier. Of course the element of social inequality appears here and may frustrate—and usually does—our attempts to guard against the mechanical result of overwork.

Patients with well compensated valvular lesions should be cautious about moving suddenly into very high altitudes; also about laboring or taking active exercise at an altitude much above that to which they are accustomed. Ill effects may not be felt at the time, but often are manifested subsequently. Some cases of valvular disease, even with beginning muscle failure, are benefited by a temporary sojourn at an elevation from one to two thousand feet higher than they have been accustomed to (mitral disease, for instance, particularly regurgitation).

During the period of compensation there is, as a rule, little or no demand for treatment. In some cases of aortic disease palpitation may be troublesome. In mitral stenosis palpitation and arrhythmia may occur from nervous excitement or from gastric or abdominal disturbance. Regulation of the diet, with the administration of intestinal antiseptics where intestinal fermentation is present, will often relieve these rhythmic disturbances. Among the most useful and innocuous antiseptics are benzosol (four grains), thymol (two grains), and charcoal (three grains), in capsule. When the rhythmic disturbance is due to cardiac excitement, ten grains of sodium bromide with one drop of tincture of aconite, three or four times daily, is very useful to quiet and regulate the heart. Strontium bromide (10 to 15 grains) may be given in place of sodium with advantage. Potassium bromide is very useful for nervous distress and restlessness incident to valvular lesions. The use of cactus, adonis, coronilla and remedies of this group

in this connection is not rational. When, however, beginning cardiac weakness is associated with disturbances of rhythm, the use of these remedies may be very satisfactory, much more so, indeed, than the use of more powerful medicines.

The diet of cardiopathic patients should be carefully regulated. This pertains especially to the hyposystolic period of heart lesions, but early care of the diet may save much trouble. Adler and Stern, from examinations made after test meals, conclude that the stomach of cardiac patients is not materially different from the normal stomach. Einhorn concludes that heart disease does not diminish the acidity of the gastric juice, and that gastric symptoms are the result of congestion of the mucosa.

Definite rules should be given cardiac patients about their manner of eating and drinking, to the effect that the meals should be taken regularly. There must not be less than five or more than six hours between meals; there must be no food taken between meals. (When marked cardiac failure is present and nourishment is important, food in concentrated form, such as peptonized milk, beef peptonoids, somatose, etc., may be given every three hours). The amount of fluid taken with the meals should not exceed eight ounces. Mineral or plain water, or seltzer water and milk may be taken between meals. Balfour recommends sipping hot water between meals as a cardiac stimulant more valuable than alcohol. The latter he justly objects to. Extremes in the temperature of ingested fluids affect the pulse rate and blood pressure. According to Strickler and Friedrick, cold water diminishes the pulse rate and raises the blood pressure; very cold water sometimes diminishes the blood pressure; warm water quickens the pulse rate and raises the blood pressure; lukewarm water usually lowers the blood pressure. These effects are more marked in diseased conditions than in health. The only alcoholic drinks that should be allowed are an occasional glass of dry wine, like Rhine wine, moselle or claret at meal time. The abuse of alcohol (and any continued use of the stronger alcoholic preparations constitutes abuse in this connection) is a most dangerous thing in cardiopathic patients. Care must also be exercised in regard to the use of tea and coffee. Stokes long ago pointed out the tendency

of these substances to produce irregularities of the heart's action. Personal idiosyncrasies are very marked, however, in relation to these drinks.

Tobacco should be altogether interdicted. Bernard showed that tobacco increased the pulse rate, and many observers have declaimed the disastrous effect of tobacco on the heart. (Decaisne observed 21 cases of intermittence of the pulse independent of organic lesion, in 88 confirmed smokers. He thinks a state of *narcotism* of the heart is developed, characterized by pulse intermittence.) The bad effect of tobacco on the cardiac rhythm is particularly evident in functional disorders where there is a tendency to rhythmic alterations. In many of these cases the digestive functions are to blame, nevertheless, in a person with well-marked functional or organic disease of the heart, the use of tobacco should be prohibited.

Mineral waters are of little therapeutic value beyond the fulfillment of some temporary indication. Beaumetz declares that they are contraindicated, especially the sulphurous waters.

The character of the food should be adapted to the individual patient with the object of easy and complete digestion. In general, the carbohydrates,—fats, sugars and starches—should be eliminated as far as consistent with maintaining a nutritious diet. Meats and meat preparations may be allowed providing the digestion is fair and the kidneys are not involved in the case. For cases of marked ataxia of the heart, milk, buttermilk, or milk and seltzer water is the best diet. Huchard especially commends milk diet for the disturbances arising from the auto-intoxication of renal incompetence. Moreover, milk is a valuable diuretic, which fact, according to Sée, is due to the lactose contained therein. In cases where dropsy is present and a mixed diet can be taken, a diet as dry as possible is recommended for its favorable effect on the œdema. In my experience the good effect of this diet is not sufficient to balance the difficulty of maintaining it.

In some cases the first evidences of insufficiency or arrhythmia, or both, on the part of the heart are connected with some disturbance of the alimentary tract, and regulation of this will correct the cardiac symptoms. Many of the disagreeable symptoms of a weak but not failing heart, which

arise from visceral congestion, are relieved by attention to the
gastric and hepatic functions.   Indeed, this is true of the most
advanced cardiopathies also.   It is wonderful what a change
for the better is often induced in a weak circulation by the
administration of a few doses of mercury.   Murry says that
mercury, in the form of blue pill, is extremely valuable in
some cases of heart disease.   A useful combination is the fol-
lowing:

R

Irisin,   gr. $\frac{1}{3}$.
Euonymin,   gr. $\frac{1}{2}$.
Ext. nucis vom.,   gr. $\frac{1}{6}$.
Hydrarg. mass.,   gr. 3.

M. S.   For one pill to be taken at bed time.

In some cases four or five quarter-grain doses of calomel
may be preferable.   In many cases of cardiopathies after
middle life the continued administration of small doses of mer-
cury is decidedly beneficial.   Here the red iodide, or the cor-
rosive chloride, of mercury, may be used.   Morrison says that
the virtues of mercury are conspicuous in cardiac dropsy with
hepatic congestion.   Beatty says that mercury is of value in
venous engorgement from chronic, primary mitral disease with
back pressure; in venous engorgement from mitral incompe-
tence secondary to old-standing aortic disease; in dilatation
with general dropsy, but no murmur; in venous engorgement
from failure of the right heart in emphysema or bronchitis;
and in venous engorgement in the late dilatation associated
with Bright's disease.   One-half grain of calomel may be given
in pill with digitalis and squill every four hours night and day
for ten or fourteen days.   If diarrhœa should appear, change
to a similar pill containing in addition one-eighth to one-half
grain of powdered opium.   W. H. Thompson says that in
chronic endocarditis good results are often obtained from 1-24
of a grain of perchloride of mercury given thrice daily for a
week.   Wood points out that with hepatic congestion, œdema,
and a hard pulse, digitalis acts poorly, and mercurial purges
are necessary.   He also advises from 1-100 to 1-50 of a grain
of corrosive sublimate with tincture of the chloride of iron for

continued administration. Murry cites the case of a man with chronic cardiac disease, who took 20,000 grains of blue pill in ten years with great benefit, and without salivation, nausea or purgation.

Moral, as well as physical, hygiene must be considered in managing cardiopathies. Strong emotions and passions affect the heart disastrously. Certain restrictions must therefore be placed upon the passions and emotions. Another question with hygienic relations is that of the marriage of cardiopathic patients. While such patients are not at all likely to follow advice in this matter, even after having asked it apparently in all sincerity, we should place the facts before them and let them assume the responsibility. As a rule pregnancy affects unfavorably any valvular lesion. Dangerous symptoms are likely to appear during gestation, or during, or immediately after, delivery. The dangers to the woman are, from hæmorrhages, syncope, rupture of the heart, or thrombus of the heart. Miscarriages are frequent (Durosier found 21 abortions or premature births in 41 patients). Birth is apt to occur at seven months. The children die early (37 out of 40 children from cardiopathic mothers died before the sixth year—Durosier). The greatest danger to the mother is at about seven and a half months. Chloroform should be used during labor, and the woman should be saved violent effort by every proper means at the command of the accoucheur. The mother should not be allowed to nurse the child. In a pregnant woman, with chronic endocarditis, if compensation be perfect and the kidneys are healthy, she will probably go through her gestation and confinement without trouble. If, however, she has suffered from chorea in early life, one may expect trouble from this source between the sixth and eighth months of gestation, especially in a primipara. According to McDonald, aortic insufficiency and mitral stenosis are the most unfavorable lesions in relation to pregnancy. Sée says that mitral stenosis is less dangerous than regurgitation as far as labor itself is concerned. In relation to abortion, mitral insufficiency is said to be the most unfavorable, and aortic insufficiency the mildest lesion. Marked mitral stenosis is the most disastrous of all valvular lesions (Handfield—Jones). In a primipara with an

aortic valvulitis and an early history of chorea, the latter trouble reappeared at the sixth month of gestation, was uncontrolable (speech and deglutition becoming impossible) and premature labor was induced at seven and a half months. Delivery was accomplished with difficulty, and the patient recovered without any appreciable ill effect on the heart.

There are some medicinal measures which, while applicable to any period of cardiopathies, find their chief clinical application in the threatened failure of the heart which antedates actual dynamical inability on the part of the heart muscle. Such remedies as iron, strychnia, arsenic, the iodides and mercury are of most use at this time. Balfour recommends in old people the persevering use of from four to six drops of a clear mixture of equal parts of liquor strychniæ hydrochloratis and of liquor arsenici hydrochlorici. Iron in all anæmic patients; in young persons the tasteless tincture is an available preparation; in older people the liq. ferri et am. acetatis (Basham's mixture), or the iodide of iron are useful. Strychnia (1-30 gr. t. i. d.) is a valuable tonic for continued administration. In cases where cardiac energy is defective without evident structural lesion, strychnia may be given continuously for years with benefit (Balfour). Iodide of sodium is preferable to the potassium salt. It may be given in doses of three to five grains thrice daily, and is useful for continuous administration in adult life because of its tendency to oppose vascular rigidity. Arsenic is useful (Fowler's solution) when valvular disease is associated with vascular sclerosis. Mercury may be employed in the manner already spoken of.

When the patient is seen early enough the careful use of the measures already alluded to may postpone indefinitely the actual development of muscle failure. The majority of patients, however, have already entered upon the hyposystolic period of chronic endocarditis when they first appear for advice. They demand remedies more potent for immediate results than are those already spoken of. It is now simply a question of the integrity of the heart muscle and the urgency of the demand for help. In general, the indications of a failing muscle are: changes in the pulse, dyspnœa, œdema, renal insufficiency, visceral congestions; as of the lungs, liver and

stomach. Actually, we judge of the condition of the heart muscle by the quality and character of the first sound as related to the second sound, and by the nature of the physical changes in the heart; by the character of the second pulmonic sound in mitral lesions, and of the aortic second sound in aortic valvulitis in relation to the vascular tension. We must remember that muscle failure may be, in a sense, relative to, and dependent upon, increase in the peripheral resistance. Thus, in a woman of sixty years, presenting to a distressing degree all the symptoms of cardiac weakness in connection with sclerosed vessels, marked relief and comparative comfort for a long time was obtained through five drops of tincture of opium thrice daily.

Right here we may mention the necessity, in most cases, of the exhibition of a vaso-dilator in connection with cardiac stimulants, especially with digitalis, which contracts the arterioles. Particularly in persons above middle life a vaso-dilator is necessary if we are to get the best effect from such a drug as digitalis, and many failures with the drug are due to the omission of this very necessary adjunct. The best vaso-dilators are opium, nitrite of sodium and nitro-glycerine. Opium is the best because it is the safest, most reliable, and the dose is most easily adjusted. From two to five drops of the deodorized tincture of opium may be given with the liquid preparations of digitalis, or one-third of a grain of powdered opium may be used with the solid preparations of digitalis. Chemically pure nitrite of sodium is a reliable vaso-dilator, the dose, however, is more difficult to adjust as some persons are very sensitive to its action. From one to five grains may be given thrice daily. Nitro-glycerine is a powerful vaso-dilator. For this purpose it should not be given in doses large enough to stimulate the heart. One two-hundredth of a grain (in tablet form) will answer. Its action is much more rapid and transient than that of opium or sodium.

If pronounced symptoms of failure of the heart muscle are present, rest in the recumbent position is necessary. The more absolute the rest, the more pronounced the benefit. By rest we relieve the excessive intraventricular pressure which is slowly but surely dilating the ventricles, and the medicaments

have greater possibilities for good.   Marked improvement will
often be obtained through rest alone, without any medication.
Muscular exercise should be given by massage, during the rest
cure, or, in suitable cases, passive or resistance movements may
be utilized.

In connection with the medicinal treatment of the hypo-
systolic period of cardiopathies we will consider first, those
medicaments which are used to increase the dynamical power
of the heart muscle.

Digitalis.   The medicinal treatment of the hyposystolic
period of cardiac lesions is the most important feature of the
therapeutics of this stage, and the most important remedy is
digitalis.   It is unnecessary to consider here the origin, history,
or mode of action of digitalis.   It is the indications for its use
and its practical application in which we are interested.   The
one cardinal indication for the employment of digitalis is failure
of the heart as a pump.   Digitalis is indicated in any case where
there is dynamical failure of the heart muscle, without refer-
ence to the nature or location of the primary valvular lesion.
There is no exception to this rule in the abstract, though there
may be individual contraindications, such as intolerance of the
drug, or a dangerous degree of muscular degeneration.   With-
out wishing to discuss the question of the essential nature of
the action of digitalis, we may note that the majority of car-
diopathies appear for treatment with well marked dynamic
failure.   We stimulate the heart to more energetic contraction
with digitalis.   If the integrity of the muscle be fair, the slower
and firmer contraction determines better blood supply to the
muscle, and thus the tonic action of digitalis in building up the
tone and integrity of the muscle is instituted and may be
of permanent benefit if the drug is properly handled.   On the
other hand, if the muscle is sufficiently degenerated, we will get
a modified and temporary stimulant action from the digitalis,
which, after a few days of unsatisfactory struggle on the part
of the heart, is lost, and the latter relapses into a condition of
asystolism.   Thus we say that clinically—in the beginning of
a case—we employ digitalis most often as a stimulant, and the
tonic properties are dependent on its virtues in this regard.
Therefore it should be used at first, as any other stimulant or

symptomatic medicine is used, in such doses as will produce the desired effect. Balfour says that digitalis is used for these purposes: first, to improve the nutrition of the myocardium and thus augment the force of the contraction; second, to contract dilated ventricles; and third, to remove dropsy. I would re-state the first proposition in this way: We give digitalis to augment muscular contractility and thus to promote nutrition of the myocardium, for, better nutrition postulates better blood supply to the muscle, which, in turn, postulates more forcible cardiac contraction. If we did not get the latter we could not obtain the former as a result of the administration of digitalis. These results obtained, we can prolong the effect possibly for years by much smaller and continued dosage. Ewart cautions against digitalis in stenosis, either of the periphery or at the center, and says that dilatation of the heart and of the orifices is the indication for its use; while advanced atheroma, partic-ularly cerebral, constitutes a major objection.

During the earlier history of the hyposystolic period of aortic stenosis, digitalis is often not as serviceable as strophan-thus, for the arrhythmia so often a feature of this lesion seems at times to be increased rather than diminished by digitalis. When right ventricular failure is present, however, digitalis is of service. Much objection has been made to the use of digi-talis in aortic regurgitation. Considering that, mechanically, aortic regurgitation presents the most difficult conditions to manage of all the valvular lesions, and that the ventricular dilatation is a progressive affair which can never be brought to a standstill unless the leak be slight, we find that digitalis is relatively as serviceable in cases of aortic regurgitation as in other valvular lesions. The dose, however, should be from one-quarter to one-half greater than in mitral lesions. A man came under observation with aortic regurgitation. He had been taking for several years with more or less regularity, on his own responsibility, doses of from one to two drams of tinc-ture of digitalis, once, twice, or three times daily, or as he expressed it, a mouthful when he thought he felt the need of it. The dose was reduced and the heart acted badly. Death occurred shortly afterward, probably from cerebral embolism. Balfour does not agree with the statement that the action of

digitalis is generally bad in aortic regurgitation. He thinks digitalis is indicated in any case no matter what the nature or cause of the concomitant lesion may be. According to Morrison, digitalis is useless in aortic regurgitation, and belladonna, nitrites or strychnia are indicated to quicken the circulation; the circulation being largely due to the aspiration force exerted by the ventricle and by the inspiratory act. Digitalis is indicated in mitral lesions where maximum aspiratory force is required.

The most reliable preparation of digitalis is the infusion made from the fresh English leaf (a cultivated leaf free from stems and stalks which contain, according to Brœker, only 20 per cent. as much digitalis as is present in the leaf). It is more eligible than the leaf itself. The active principles of digitalis are, it is claimed by chemists: digitalin, almost insoluble; digitoxin, wholly insoluble in water; digitonin and digitallein, both fully soluble in water. The fluid extract and the tincture probably contain all of these principles, but not in constant proportions. The infusion contains digitonin and digitalein with but a trace of digitalin, and is said to be free from digitoxin which is the most active and dangerous constituent of digitalis. The infusion is therefore the safest preparation and its dose is the most easily graded. A useful formula is the following:

R

Tinct. opii deodor.,	ʒi-ii.
Kalii nitratis,	ʒiv.
Inf. digitalis (fresh English leaf) q. s. ad.,	℥xii.

M. S. Tablespoonful once in four to six hours.

The powdered leaf is preferred by some, and is probably the most reliable of the solid preparations. The dose of digitalis is more or less arbitrary and is stated about as follows: Maximum dose of German digitalin, 1-16 of a grain; French digitalin, 1-32 of a grain; digitoxin, (the most active, definite and poisonous preparation of digitalis), 1-64 of a grain. The maximum dose of digitalis leaves has been given as five grains, toxic dose ten grains. The dose of any preparation of digitalis will necessarily vary so much under different circumstances

that fixed dosage has no place in the therapeutic application of the drug in cardiopathies. Arnold and Wood Jr., from an experimental comparative study of digitalis and its derivatives, came to the following conclusions: 1. Digitalin and digitoxin each represent the full circulatory powers of digitalis. 2. Digitalis, digitalin, and digitoxin stimulate the cardiac-inhibitory mechanism both centrally and peripherally. In larger doses they paralyze the intrinsic cardiac-inhibitory apparatus. 3. They all cause a rise in blood-pressure by stimulating the heart and constricting the blood vessels. 4. Very large doses paralyze the heart muscle of the mammal, the organ stopping in diastole. 5. Digitalin of Merck is a stable compound, one gm. of it being equivalent to about 70 cc. (18 dr.) of tincture of digitalis. 6. Digitoxin is not recommended for human medication on account of its irritant action, which makes it liable to upset the stomach when given by the mouth, or to cause abscesses when given hypodermically, and on account of its insolubility, which renders it slowly absorbed and irregularly eliminated, having a marked tendency to cumulative action.

We find our cases in a condition of ruptured compensation. We at once set about to correct the irregular circulation by comparatively large doses of digitalis, grading the dose by the effect. We do not stop to grade the dose so that the quantity of drug ingested is balanced by that excreted, before a second dose is administered, by which the tonic action of the drug is attained (Balfour). But, having attained a restitution of the circulation, the latter method of dosage (one grain of powdered leaves or its equivalent in twenty-four hours, or 1-100 grain doses of digitalin—(Balfour) is adapted to maintaining the effect. We would unquestionably have more permanent results if we could maintain for years a more or less constant administration of small quantities of digitalis, instead of losing sight of our patients as soon as the circulation is restored, not to be seen again until dilatation has again attained a dangerous degree.

Beates has recommended digitalin (German, pure— Merck) in doses of from 1/10 to ½ of a grain, as a reliable cardiac stimulant. This apparently large dosage is effective and is

well borne by the stomach. Beates concludes that: 1, Digitalin (German, pure,—Merck) is a derivative of digitalis not contaminated by other active principles; 2, It possesses a uniform and unvarying strength; 3, It is relatively free from the property which produces gastric irritation; 4, It is a powerful stimulant to the whole cardiac apparatus; 5, It is a pronounced and reliable stimulant to the vaso-motor system; 6, It does not develop cumulative action; 7, Adult dose from 1/10 grain (minimum) to $\frac{1}{2}$ grain (maximum). In two women, seventy-two and sixty-five years old respectively, one with an atheromatous aortic stenosis, and the other with a rheumatic aortic lesion with secondary mitral leakage, digitalis in one-tenth grain doses gave excellent results without any evidence of gastric disturbance. A good formula is:

R

Digitalin,	gr. 1/10.
Strychnia sulph.,	gr. 1/25.
Pulv. opii.,	gr. 1/3.

M.
Ft. capsul no. i.

The cumulative action of digitalis, about which much caution has been given, is seldom or never observed. To suitable cases continuous administration for months is not productive of trouble, though it is better to intermit the exhibition of the drug every two or three weeks, for a few days. The asystolism which digitalis is said to develop is largely due to its administration in cases unsuitable to the drug. W. H. Thompson emphasizes the value of resting the heart from the stronger stimulants and giving aconite in acute exacerbations of chronic diseases. Aconite is indicated instead of digitalis when there is a strong, laboring heart with rapid pulse, or a quick pulse associated with intrinsic cardiac pain. A quick pulse with high tension indicates renal complication, and calls for veratrum viride, which slows the heart and dilates the arterioles.

Strophanthus. According to Fraser the action of strophanthus is mainly on the heart muscle and only slightly

on the arteries, which he considers advantageous, as it un-
doubtedly is under some circumstances. A solution of
strophanthin, 1-3,000, passed through the vessels of a frog's
web and caused no change, only slight transitory change re-
sulting from a solution of 1-2,000. A solution of digitalin,
1-20,000 caused sufficient contraction to stop the passage of
the solution.

Balfour considers the lack of action on the arterioles of
doubtful advantage, as the blood pressure is more variable and
metabolism uncertain. Strophanthin is more soluble and ab-
sorbable than digitalin, is rapid in action, and available for
hypodermic use. Strophanthus is the only member of the
group which rivals digitalis, but it is not equal to, and will not
displace, digitalis.

Strophanthus increases the force and duration of the car-
diac systole. Its tonic action is slight or altogether wanting,
as the tonicity of the heart muscle is not maintained after the
cessation of the drug, but relapses into its former condition
unless improvement in this respect has been obtained through
rest, diet, tonic medication, etc., during the period of stimula-
tion by the strophanthus. In the minor degrees of ataxia of
the heart exhibited by nervous, irritable, excitable persons,
strophanthus is particularly useful. This is especially true of
mitral stenosis, in which lesion strophanthus is particularly
effective in controlling the distressing palpitation and dyspnœa
so frequently present. In the more marked degrees of cardiac
weakness which border on asystolism, resulting from any
lesion whatever, strophanthus is not effective in stimulating
the heart to its usual dynamical condition. In some instances
strophanthus causes gastric distress and diarrhœa. Gastric
distress is not so likely to occur if strophanthus be adminis-
tered in laurel-cherry water. The dose of tincture (P. D. &
Co's.) is from seven to twelve drops. The dose of strophan-
thin is from 1/200 to 1/60 of a grain. The former dose is
large enough for hypodermic use. Strophanthin is as reliable
as other alkaloids of its class, and fairly represents the action
of the tincture. Wilcox says the gastro-intestinal disturbance
from strophanthus is due to the character of the preparation
and is independent of the amount of strophanthin. He thinks

that strophanthus is superior to digitalis in: 1. Greater rapidity of action; 2. Absence of cumulative effect; 3. Non-interference with caliber of the arteries. Strophanthus has been regarded by some as a reliable diuretic, but its action in this regard is decidedly uncertain.

Caffein. This drug has been recommended by Dujardin Beaumetz, Huchard and Semmola in asystolic conditions, arrhythmia and cardiopathies of bulbar origin, (a condition observed in bulbar affections, characterized by palpitation, dyspnœa, weakness of the cardiac sounds, œdema and dropsy, without organic disease of the heart—paralytic ataxia of the heart), as a cardiac tonic and diuretic. It is, in some few instances, an efficient cardiac stimulant and diuretic. It seems particularly adapted to those cardiopathies which are associated with vascular disease or with nephritis, or both. In asystolism which is purely the sequence of a valvulitis, caffein is not usually effective as a stimulant to the heart, though it may be as a diuretic. In too large doses it causes dizziness, faintness and irregular pulse. Caffeine is condemned by Zenetz, who thinks it is a poison acting on the spinal cord and striated and cardiac muscles; that it is very slow of excretion, raises blood pressure steadily, and in cardiac and renal patients may end in cardiac tetanus. Huchard believes caffeine to be superior to digitalis in both renal and cardiac disease. It should be given freshly prepared, after the following formula:

℞

      Caffein (alkaloid),
                       aa. ʒii—iii.
      Natrii benzoatis,
      Aq. destil. q. s. ad.,   ʒiii.
M.   S.   Teaspoonful every four to six hours.

Strychnia. This drug, next to digitalis, is the most useful of cardiac remedies. During the hyposystolic period of cardiopathies it is used as a stimulant, and is the only one which can be safely and advantageously combined with digitalis in all lesions and in all stages. It is particularly efficient as a stimulant to the right heart, and is therefore indicated in

the systolic period of all lesions. The dose as a stimulant should be from one twenty-fifth to one fifteenth of a grain. The frequent hypodermic use of strychnia is a powerful cardiac stimulant in emergencies. In doses of one twenty-fifth of a grain, thrice daily, strychnia is a useful adjuvant to the action of digitalis.

Spartein. Sulphate of spartein is a remedy of value as a regulator of functional or valvular arrhythmia. According to Voight it is indicated in weakness of the right heart. Thomas, and Cushing and Mathews agree that spartein acts directly on the myocardium. Chapman has found spartein of value in dilatation of the heart, and in dropsy from dilatation of the right ventricle. The dose being from $\frac{1}{2}$ to 1 grain every four hours. Its action is rapid, and it may be used to sustain the heart until digitalis has had time to produce its effect. Spartein will not fill the place of digitalis in cases of actual muscular failure. It is of use in cardiac weakness and irregularities in neurotic individuals. It is also serviceable to steady the pulse in persons addicted to the opium or other drug habits. The dose of sulphate of spartein is from one-half to two grains. It is readily soluble and can be given hypodermically if desired. Thomson recommends the following pill when digitalis is not well borne:

R

Sparteinæ sulphatis,	gr. i.
Pulv. scillæ,	gr. ss.
Caffeinæ citratis,	gr. iss.
Strychniæ sulphatis,	gr. 1/30.

M. Ft. Pil. No. i.

Convallaria. Convallaria majalis was used as early as the sixteenth century for diseases of the heart. According to Sée it augments vascular pressure and the ventricular energy, is useful in palpitations and arrhythmia even if due to valvular lesions; its diuretic power is great and it is indicated in all cardiopathies attended with dropsy. It is also recommended in the various irregularities and conditions arising from mitral disease. The dose of the officinal fluid extract is from 15 to

30 minims. The active principle of the drug having cardiac action is *convallamarin,* the dose of which is from one-fourth to one and one-half grains.

Cactus Grandiflorus. (Fl. ext., 5 to 10 minims; tincture, 15 to 20 minims.) Has been specially recommended in myocarditis, angina pectoris, and cardiac arrhythmia and weakness following acute fevers. According to Wilcox it should not be given in mitral stenosis. It has no cumulative effect.

Adonis Vernalis. (Adonidin, 1/20 to $\frac{1}{4}$ of a grain.) Has the same therapeutic indications as cactus. Rummo and Ferranini claim that spartein and caffein affect the cardiac nerves and muscles little or not at all; Adonidin and convallamarin affect both the nerves and muscles simultaneously; while digitalis, strophanthus and their alkaloids affect the muscle primarily and the nerves secondarily.

Nerium Oleander. This drug has been employed by Œfele, and *Coronilla scorpioides* has been introduced by Cardot as cardiac remedies. Poulet highly recommends coronilla ($\frac{1}{2}$ to 1 dram doses of the tincture). It is useful in nervous debilitated subjects, in various functional disturbances, aids digestion, and may be useful where digitalis is harmful or has failed. These remedies are neither better nor worse than the others of their class, not any of which can be relied upon to sustain a heart muscle which has developed serious dynamical failure.

The foregoing medicaments have been considered in a group with reference to their action on the cardiac muscle. We may now refer to the symptomatic management of cardiopathies, for notwithstanding the use of the foregoing remedies, which of necessity must, if effective, relieve the various symptoms, we are often compelled to resort to special symptomatic medication for the temporary relief of aggravated conditions.

Palpitation. Cardiac palpitation is very distressing at times. In association with valvular lesions it occurs most frequently with mitral stenosis and aortic regurgitation. It is often connected with recurrent attacks of endocardial rheumatism in which case it is controlled by salicylate of sodium. Palpitation in the early history of valvular lesions, if not due

to reflex disturbance from the liver or gastro-intestinal tract, may be due to derangement of the cardiac nervous mechanism. Rest is important. Cold applications (ice bags) to the præcordium or to the nape of the neck are useful, and are usually effective in proportion to the absence of arrhythmia. Bromide of potassium or sodium with small doses of aconite are useful for arrhythmic palpitations. When these means fail, small doses of opium are necessary. In the palpitation which is sometimes such a distressing feature of the asystolic period of an aortic regurgitation, opium is the only satisfactory relief. One-quarter to one-half grain of the sulphate of codeine, five to ten drops of deodorized tincture of opium, or one-eighth to one-fourth grain of the sulphate of morphine (hypodermically) may be used. Codeine and sodium bromide make an effective combination. It is well to remember that opium is, in a sense, a cardiac stimulant (Gubler), and that properly administered and watched it is, in these circumstances, a perfectly proper, safe and effective remedy.

Præcordial Pain. Pain hardly deserves mention here, as it is not an attribute of valvular lesions. Yet it may be present in aortic regurgitation or stenosis, and occasionally in mitral lesions. When excessive heart action is present, bromides, opium or cold applications may be employed. In aortic valvulitis with general vascular disease, nitrite of amyl or nitro-glycerine may be used. Liegeois recommends the tincture of piscidia erythrina for the painful sensations associated with myocarditis or endarteritis; in gouty, senile and tobacco subjects. Twenty-drop doses morning and evening were effective, though eighty drops daily may be given continuously.

Dyspnœa. The constant dyspnœa present in the advanced stage of valvular lesions, and attributed by many to left auricular failure, is, in most instances, evidence of an overtaxed right ventricle. The dilatation of the alveolar vessels actually diminish the breathing space. Nitro-glycerine is often used for this dyspnœa, and may give relief through general stimulation; the real remedy, however, is strychnia. When the dyspnœa is spasmodic in character, and is associated with vascular sclerosis, the vaso-dilators are indicated such as nitro-glycerine (1/200 grain, in tablet, three to four times daily),

nitrite of sodium (2 to 3 grains, in water, t. i. d.), codeine,, morphine, or deodorized tincture of opium. Opium is very satisfactory for dyspnœa, and will give relief while we are waiting for the action of some slower remedy. As a result of Herouet's observations he concludes, that morphine is admissible in mitral stenosis when other means fail. It calms dyspnœa and nervous symptoms and induces sleep. Heitler considers the hypodermic use of morphine and ether as the most rational treatment of severe cardiac dyspnœa,—morphine one-twentieth to one-tenth grain doses at first, with ether injections if the pulse continues small and weak.

Syncope and Cyanosis. Attacks of syncope are most likely to occur in connection with aortic stenosis or mitral stenosis. Rest in the recumbent position, with the head low, is necessary. Stimulation may be given with alcohol by the mouth or hypodermically. Camphor (hypodermic injections of camphorated oil) or musk may be given, though the latter is not very satisfactory. Forty or fifty minims of ether hypodermically is very efficient. In cyanosis, in addition to the above treatment, we should use large doses of strychnia hypodermically (1/25 to 1/16 of a grain). In dyspnœa and cyanosis associated with marked venous stasis, especially in connection with mitral stenosis, we should not hesitate to bleed. The abstraction of a few ounces of blood will not only fulfill symptomatic indications, but will save and prolong life. Pye-Smith considers cyanosis with dilatation of the right heart either from pulmonary stenosis or other obstruction, as an indication for venesection, and is corroborated by Lafleur, who says that the most benefit is obtained in cases of primary cardiac and arterial disease. According to Huchard we should bleed when, in sclerosis, the ventricle is unable to meet the increased peripheral resistance. Advanced disease of the coronary arteries or heart muscle are contraindications for bleeding.

For gastric distress from fermentation, correction of the diet should suffice; if not we may use the following:

℞

    Natrii bromidi,   ʒiv.
    Bismuth subcarb.,   ʒi.
    Pulv. carbo. lig.,   ʒii.
    Aq. cinnam. q. s. ad.,   ℨiii.
M. S. One teaspoonful every 4-6 hours.

W. H. Thompson advises, for flatulence, ten grains of sodium benzoate thrice daily and two drams of sodium phosphate in hot water in the morning, sipped slowly; or blue mass every fourth night, and half an ounce of sodium phosphate the next morning. For sluggish kidneys a copious saline rectal irrigation at 110 F. is efficient.

If moderate jaundice appears, in spite of the use of mercurials already alluded to, we may give sodium phosphate in one-half to one dram doses, three or four times daily. For diarrhœa, which is sometimes troublesome, the bismuth preparations are best, especially the sub-gallate in five to eight grain doses every three to six hours. It may be necessary to use small and continued doses of tincture of opium. If the diarrhœa is watery with frequent passages, we may give the following:

℞

    Tr. oppi deodor,   ʒii.
    Tr. camph.,   ʒii.
    Acid nitrici dil.,   ʒiii.
    Elix. simp. q. s. ad.,   ℨiij.
M. S. Teaspoonful after each stool.

Dropsy. Dropsy is an important though erratic symptom in valvular disease. It is important because it shows in a general way the imperfect circulation, and because the patient attaches immense consequence to it. If we can manage the œdema, we may be sure the patient will rest satisfied with our management of the other features of the case. Œdema may be, at first, largely a matter of gravity, and the recumbent position will often relieve it, or it will disappear with the increased vascular tension incident to the administration of

digitalis.   If the blood pressure is very low bandages to the
legs will aid the digitalis.   The infusion of digitalis should be
given and if the œdema is well marked, a brisk cathartic should
be used.   One of the best cathartics is compound jalap powder,
in dram doses morning and evening (Canfield).   In some
cases of sclerosis of the arteries with a firm, tense pulse, digi-
talis will not cause diuresis unless we accompany it with a
vaso-dilator (opium, nitro-glycerine, or other nitrites).   Digi-
talis is efficient in those cases with weak, irregular pulse, and
soft, doughy, easily pitting tissue.   At least a half an ounce
of the infusion must be given every four hours.   Again, though
we regulate the kidney circulation in one or both of these ways,
diuresis may not occur.   We will then have to add some stim-
ulant to the kidney itself.   Here the old time pill consisting of
one grain each of digitalis, squills and calomel, is useful.   Ten
or fifteen grains of the acetate, or nitrate, of potassium added
to the dose of infusion of digitalis may be efficient.   Glax ad-
vocates the treatment of cardiac dropsies by restriction of the
fluids ingested to correspond with the amount excreted.   He
thinks this method will re-establish compensatory action of the
heart.   Either a comparative or absolute dry diet is difficult
to carry out, and while it is quite possible to give too much
fluid to some cases, I do not believe that a dry diet reconciles
the physiological conditions   and   therapeutical   indications
which we have to meet.

Squills, broom tops, juniper (teaspoonful of the fluid ex-
tract thrice daily), or Trousseau's diuretic wine, are useful
though the latter is more adapted to cases associated with
nephritis where we are not giving cardiac stimulants.   The
following is the formula of Trousseau's diuretic wine:

R

Digitalis fol.,	10 gms.
Scillae rad.,	5 gms.
Bacc. juniperi,	50 gms.
Vini alb.,	750 gms.

Macerate for four days, add acetate of potash, 15 gms.
Filter.   Dose, tablespoonful three or four times in 24 hours.

Bitartrate of potassium in connection with digitalis is often of service, or the following formula, as recommended by Brunton, (*Haustus Scoparii Compositus*—St. Bartholomew's Hospital): potassii tartratis, gr. xx; spirit. juniperi, м. xxx; decocti scoparii, ad ʒi, may be used with digitalis. The old "Imperial" drink (*Potus Potasii Tartratis Acidae*), long used in fevers, may also be serviceable as well as agreeable. It may be prepared as follows: Acid tartrate of potash, 60 grains; sugar and boiling water a sufficient quantity; one or two lemons, stir occasionally till cold, strain and administer.

In severe cases of dropsy from combined renal and heart disease, caffein (natro-benzoate) after the formula already given, is decidedly diuretic. When large quantities of fluid can be given milk is a good diuretic. According to Sée the diuretic qualities of milk are due to lactose which is a certain and powerful diuretic in cardiac dropsy, lactose being better than milk diet as it admits of meat or other food. It is soluble in cold water, one part in six, and one hundred grammes daily produces marked diuresis.

Theobromine is recommended by Sée as an unfailing diuretic. It is insoluble and is given in powder (capsule) with the food or in milk. First day, $1\frac{1}{2}$ grammes; second day, 3 grammes; third day, 4 grammes; then suspend treatment for five days; then if necessary give 2 grammes daily for three days.

Our two most valuable special diuretics, however, are calomel and diuretin. Sée, Pepper and others recommend calomel as a special diuretic. It is the most reliable diuretic we have for cardiac dropsies. I have come to regard the failure of calomel in pure cardiac dropsy as due not to the drug itself, but to failure to properly adjust the circulation, or to improper administration. If the circulation be stimulated and the kidneys be not too much diseased, then calomel will surely cause diuresis. If the pulse is weak and intermittent, digitalis should be given for two or three days before giving calomel, which is then administered in three-grain doses, morning, noon and night, for two days—eighteen grains in all. During this period the bowels should not be allowed to move. (This can be effected by giving five drops of deodorized tincture of

opium half an hour after each dose of calomel.)   The diuresis
may be delayed for twelve hours after the last dose of calomel,
but usually begins on the second day of its administration.
The effect usually lasts for some time.   The effect of calomel
in nephritic dropsies is uncertain, but sometimes surprisingly
efficient.

Diuretin (sodio-salicylate of theobromine, containing, it
is said, about 60 per cent. of theobromine) is a valuable diu-
retic in cardiac dropsy.   Much clinical study has been given
the drug during the last five years.   Some believe its good
effect on the circulation is due to its diuretic properties (Paw-
inski).   Others believe it acts on the heart also (Hoffman,
Geissler).   It is decidedly doubtful if diuretin has any direct
action on the heart.   According to Brunton, diuretin increases
the excretion of water by acting on the secreting cells and
nerves of the kidneys.   Diuretin may be given in watery solu-
tion, twenty grains every four hours until six doses are taken,
—one hundred and twenty grains in all.   If this quantity does
not cause diuresis it is useless to continue it.   Less than ninety
grains in twenty-four hours is not effective.   Small and con-
tinued dosage is useless.   The effect when obtained lasts for
some days.   A patient, seen in consultation, with a most ex-
tensive dropsy, was given the calomel treatment.   Diuresis not
occurring by the time the last dose was administered, diuretin
was given.   (Sufficient time was not allowed as the action of
calomel is sometimes delayed.)   During the next three days
such an enormous quantity of water was eliminated that the
man went into a condition of collapse, with a pulse of 160 and
sighing, rapid respiration.   He was resuscitated by the rectal
injection of several pints of normal salt solution.

If we are able to adjust the circulation—and there are few
cases where we cannot stimulate the heart sufficiently to allow
of diuresis—we can almost invariably get rid of dropsical ac-
cumulations.   I have never been obliged to resort to Southey's
capillary tubes, and very rarely to tissue puncture.   When the
latter method is resorted to, it must be done with antiseptic
precautions.   The leg must be washed with warm water and
soap, then with sterilized water, then with alcohol.   A large
needle is sterilized in a flame and a number of punctures made

in the skin of the calf of the leg, twenty or thirty punctures being made in each leg at one sitting. The leg is then wrapped in a thick layer of cotton. When the cotton is saturated it is changed, and the leg washed with sterile water. The cotton may be covered with oil silk or rubber. Should inflammation of the skin develop, it can usually be allayed by a fifteen per cent. zinc oxide—lanoline ointment.

Hypnotics. Insomnia is frequently a troublesome condition in cardiopathic patients, particularly in those suffering from aortic lesions. The mental hallucinations and peculiarities so marked in some cases are always more aggravated at night. It is not always easy to decide whether to use a hypnotic or not, or as to which one we shall rely on. Various drugs have been praised for their freedom from cardiac depression and their hypnotic power. But, after trying them all we usually return to the old stand-by,—opium. A quarter of a grain of morphia, with fifteen grains of sodium bromide, is the most reliable hypnotic. Chloral and sulfonal are unsatisfactory in moderate doses, and unsafe in full doses. Trional (5-10 grain doses) and tetronal (5-15 grains) are safer than the former, but uncertain in conditions affecting the intracranial circulation. Chloralamid (30-40 grains) is relatively safe and efficient. In large doses it has some disorganizing action on the red blood cells. Paraldehyde ($\frac{1}{2}$ to 1 dram) has gained ground as a hypnotic. According to Coudray, it does not depress the heart or lungs. It may be administered after the following formula:

R

Paraldehydi,	ʒi.
Spirit. chloroformi,	M xv.
P. tragacanth,	Ɔi.
Syr. auranti cort.,	℥ss.
Aqua q. s. ad,	℥iii.

M. S. One or two doses.

These are the best of the newer hypnotics for use in this connection. Their action is, however, uncertain. Some act better if given in conjunction with a small dose of morphine,

though better results will be obtained if the latter with sodium bromide is depended on.

The medicinal treatment of the hyposystolic period of cardiopathies is often disappointing. However, it still maintains its place as the principal method of managing the pronounced degrees of cardiac failure. The mechanical methods of treatment which can be so successfully applied to many cases of moderately developed ataxia of the heart are not suitable for use in the more advanced conditions because the heart is unable to stand the initial rise in vascular pressure which attends the employment of all varieties of mechanical therapeutics. For the same reason these methods are contraindicated in acute simple dilatations of the heart from heart-strain or from rapidly developed muscular weakness in acute febrile diseases. It is true that in the latter condition the more powerful cardiac stimulants are open to the same objections though not in so positive a way. Rest and medicinal measures are, therefore, our main reliance in any condition of muscular failure when the weakness of the muscle, either actually or relatively, is too great to allow of even temporary increase in resistance, the mechanical measures being admissible only when there is a certain degree of dynamic power of the heart muscle with a tendency toward compensatory development. The proper employment of mechanical therapeutics of the heart postulates a nice discrimination as to the relative capacity of the heart cavities and the power of the heart muscle, involving accurate diagnostic and prognostic observation as to conditions of dilatation and hypertrophy which we will consider hereafter.

CHAPTER VI.

ENLARGEMENT OF THE HEART.

Under the general designation of enlargement of the heart, we include those alterations in the physical conformation of the heart known as *hypertrophy* and *dilatation.* Neither of these conditions is to be considered as a disease *per se,* but rather as a mechanical result of altered dynamical relations between the heart muscle and the intracardiac blood pressure. Various classifications have been made of cardiac enlargements. Fraentzel has classified as *idiopathic,* those enlargements of the heart which have no ætiological relations to valvular lesions.

[This specious distinction is really taking *pars pro toto,* for, taking the term *idiopathic* in its generic sense, as ætiologically connected with cardiac enlargements as a physical result of dynamic causes, and not as related to primary causes which have instituted the sequence of events terminating in enlargement of the heart, it would be better to apply the term *idiopathic*—if used at all in this connection—to those enlargements of the heart arising from causes situated within the heart itself, whether these causes be situated at the valves or are *intra muros.* Those enlargements of the heart which are due to alterations in the blood pressure in the aortic or pulmonic system of vessels, are, as far as their ætiological relations are concerned, *exocardiac* in nature. Cardiac enlargement occurs because of increased intracardiac blood pressure. (This may be from actual increase in general blood pressure or may be a relative increase from diminished dynamical power of the heart muscle.) Whether the enlargement be from dilatation, or hypertrophy, depends upon two factors; first, the rapidity and extent of development of the increase in

pressure; second, the integrity of the heart muscle. If the increase in pressure is decided and rapidly developed, then dilatation will result. *Per contra*, if the increase in pressure is developed slowly and the heart muscle has not undergone degeneration, then hypertrophy will occur.]

While cardiac hypertrophy and cardiac dilatation are not in themselves pathological entities, their diagnostic, prognostic and therapeutical relations are of such importance as to necessitate the most careful consideration.

CARDIAC HYPERTROPHY.

Cardiac hypertrophy, in so far as it may occur, represents a more or less successful effort at cure on the part of nature. The term "excessive cardiac hypertrophy," is misleading. Cardiac hypertrophy occurs as a process purely compensatory for an abnormal resistance to the emptying of the heart cavities. The heart muscle does not hypertrophy in excess of the demand for power, and except in the relative sense of diminution in demand for power after that power has developed (in athletes for instance, who drop their physical work after long period of training), we are not confronted with the necessity of modifying excessive hypertrophy. [The excitable, forcible cardiac action which is often present in cardiopathies where hypertrophy has developed, is not due to the degree of hypertrophy but to disturbance of the cardiac, vaso-motor or general nervous systems. The remedies which are successfully employed in relieving such irritable hearts, are those that modify these nervous disturbances and they could not have any effect on the degree of hypertrophy.]

Cardiac hypertrophy has been divided into simple, (as used here the term has no reference to the histology of hypertrophy), eccentric, and centripetal or concentric (Boulland).

Simple hypertrophy (hypertrophy without increase in the size of the cavity whose wall is involved) probably never occurs without some increase in the capacity of the cavity concerned, yet this is so slight in many instances, that the term simple hypertrophy is appropriate in a clinical sense. Eccen-

tric hypertrophy (hypertrophy with increase in the size of the cavity whose wall is involved) indicates a condition of combined hypertrophy and dilatation where the hypertrophy predominates (compensation), in contradistinction to hypertrophic dilatation in which dilatation is a predominant factor. Concentric hypertrophy (hypertrophy with diminution in the size of the cavity whose wall is involved) is of doubtful occurrence. It is difficult to see how the mechanical conditions which result in hypertrophy can induce a centripetal hypertrophy which is anything more than relatively concentric as compared to the increased volume of muscle.

Hypertrophy of the heart may affect a portion or the whole of the cardiac wall. The ventricular walls are more often affected than the auricular, and the left side of the heart more often than the right. Moderate degrees of cardiac hypertrophy may disappear with cessation of resistance to the heart's action.

ÆTIOLOGY.—The causes of cardiac hypertrophy are numerous. Eccentric hypertrophy arises in compensation for cardiac dilatation in insufficiency of the valves, adhesive pericarditis, the various forms and degrees of myocarditis, the cardiac degenerations (especially granular degeneration associated with parenchymatous nephritis), and in sudden and maintained increase in blood pressure (as in certain degrees of mechanical heart strain). Simple hypertrophy occurs in compensation for mechanical obstruction in valvular stenosis, in slowly developed and maintained increase in vascular pressure from general arterial sclerosis or atheroma. Traube, who first indicated the dependence of cardiac enlargement on arterial sclerosis, modified his views because some well marked cases of vascular sclerosis are not accompanied by cardiac enlargement. He finally regarded both conditions as due to one cause. Fraentzel prefers to regard this class of cases as due to overwork or to excessive consumption of food and drink, these causes acting on the heart through increased blood pressure. Eccentric hypertrophy may compensate for dilatation occurring as a result of a weakened myocardium from insufficient blood supply caused by vascular disease not necessarily attended with high blood pressure. Atheroma of the aorta,

aneurism, pressure on the vessels from tumors, congenital narrowing or unusual large size of the aorta, chronic interstitial nephritis, prolonged and severe muscular training, and pregnancy may also cause simple cardiac hypertrophy. French authors have generally asserted the occurrence of cardiac hypertrophy in pregnancy. Gerhardt, Loehlein, and other Germans have questioned this fact. Handfield Jones, whose researches regarding pregnancy and heart disease are well known, says that both clinical evidence and logical deduction justify us in believing that hypertrophy of the left ventricle occurs in normal pregnancy. Simple hypertrophy of the right ventricle may arise from increased pressure in the pulmonary circuit due to pressure from mediastinal tumors or enlarged bronchial glands, to emphysema, chronic bronchitis, fibroid phthisis, chronic pneumonia, adhesive pleurisy, mitral lesions, and also from deformities of the chest or spine. It is claimed that the rapid heart action induced by prolonged mental strain or excitement or the use of tobacco or alcohol will produce cardiac hypertrophy. Strictly speaking this is not a fact, for the rapid heart resulting from these influences may exist for long periods of time without any evidence of cardiac hypertrophy. It is not rapid action which induces hypertrophy, but the increased resistance to the action of the heart. If, however, the conditions which produce rapid heart action interfere with the nutrition of the heart muscle, as they frequently do, then some degree of dilatation will obtain, which is compensated for by more or less eccentric hypertrophy. In this way eccentric hypertrophy develops in compensation for the dilatation which may attend exophthalmic goitre.

MORBID ANATOMY.—Hypertrophy of the heart is a compensating hypertrophy, and as such, is a physiological process instituted to overcome resistance to the performance of function. Uniform hypertrophy—such as occurs in adherent pericardium—causes increase in the general dimensions of the organ—both internal and external—with increased thickness of the walls. The heart may attain a weight of thirty or more ounces. Hypertrophy of the left ventricle causes elongation of the heart. The septum and left coronary artery are displaced to the right, the apex is formed entirely from the wall of the

left ventricle whose wall is much thicker than normal (from $1\frac{1}{2}$ inches to 2 inches thick). In hypertrophy of the right ventricle the heart assumes a quadrilateral shape with the anterior surface consisting almost entirely of the wall of the right ventricle. The two ventricles share about alike in the formation of the apex, and the wall of the right ventricle may almost equal the left in thickness. It never fully does so, however, except in some instances of congenital disease. The auricles seldom undergo much hypertrophy, though in tricuspid stenosis the right auricle may develop considerable hypertrophy. In eccentric hypertrophy the papillary muscles and the septum may be much thickened. In hypertrophy of the right ventricle the calumnæ carneæ become much thickened and enlarged. The tissue of an hypertrophied right ventricle is usually much tougher than that of the left ventricle. On section of an hypertrophied heart, the walls are stiff and do not collapse.

There is no clinical history of cardiac hypertrophy. The subjective symptoms which occasionally present in cases of simple hypertrophy of the heart are due either to the same cause as the hypertrophy or to reflex causes.

SYMPTOMS AND DIAGNOSIS.—There are no subjective signs which are specially indicative of cardiac hypertrophy. In erethism associated with simple hypertrophy there may be dizziness, or a sense of fullness in the head, consciousness of the heart's action, and a relative sense of dyspnœa. These symptoms may also be present in eccentric hypertrophy, under like circumstances, or when compensation begins to fail. In the latter instance, however, we are really dealing with cardiac dilatation. In most cases the clinical diagnosis resolves itself into determining whether hypertrophy or dilatation be the dominant factor in the condition of the heart.

What degree of cardiac hypertrophy are we capable of recognizing by physical examination? This is a mooted question as far as authorities are concerned, yet, if we separate carefully the conditions of hypertrophy and dilatation, we must recognize the fact that any enlargement of the heart which is easily demonstrable is indicative of dilatation of the heart cavities. We must agree with Fraentzel that an increase of half a centimetre in the thickness of the cardiac wall, while

constituting a decided hypertrophy, is very difficult of demonstration. Clinically, we are not able to draw as fine a distinction between hypertrophy and dilatation, as we do in an anatomico-pathological sense, and from both clinical and therapeutical points of view we must class as hypertrophic those enlargements of the heart in which hypertrophy is the dominant feature of the changes which render the case easy of demonstration.

Traube first emphasized the importance of high arterial tension, intensification of the second heart sound, and increased force of apex impulse as a basis for the diagnosis of hypertrophy. These symptoms furnish only presumptive evidence however, and may not be present in simple hypertrophy when arterial tension has been reduced.

The pulse in simple hypertrophy is full, strong, hard, well sustained and regular. In eccentric hypertrophy it is fuller and strong, but not as hard, well sustained or as regular as in simple hypertrophy. In hypertrophy of the right ventricle alone, the pulse may be weak and irregular.

Inspection in simple hypertrophy may show more indefinite apex beat than normal if the apex is fairly under the sixth rib. If the right side is involved there may be some epigastric impulse. In eccentric hypertrophy the area of apex impulse is increased toward the left and *downward*. If the right side is involved there will be epigastric motion. By palpation we recognize the quick, direct apex impulse of hypertrophy, in distinction from the heaving, undulating, prolonged impulse of cardiac dilatation. On percussion the area of cardiac dullness will be increased laterally and downward. In simple hypertrophy this may not be demonstrable. Fraentzel says that "percussion of the heart permits of no conclusion with regard to hypertrophy." He criticises English and American physicians for their failure to separate hypertrophy from dilatation in relation to diagnosis. This objection may be just in a pathological sense, but a clinician cannot observe pathological distinctions which can have no clinical recognition. In respect to the intimate clinical association of hypertrophy and dilatation, the only practical course is to credit each condition with those physical signs which obtain while that state constitutes the dominant factor of the individual case.

In eccentric hypertrophy the area of dullness will be increased to the right or left according to the side involved. Emphysema or other intrathoracic conditions which change the relation of the heart to the anterior border of the lung may modify the area of cardiac dullness. The amount of lateral displacement of the left border of dullness, as compared with the extent of downward displacement of the apex beat, is important in estimating the relative extent of dilatation and hypertrophy. The former condition is indicative of dilatation; the latter of hypertrophy.

The first heart sound (in the absence of a murmur) will be dull, heavy, and prolonged apparently at the expense of the short interval. The second heart sound is short, loud and high pitched. (The aortic second sound is intensified in left ventricular hypertrophy, the pulmonic second sound in right ventricular hypertrophy, and both sounds in general hypertrophy.) Absence of respiratory murmur over a greater præcordial area than normal, will be apparent in the absence of emphysema. In some instances pleuritic adhesions or sclerosis of the lungs, or both, may operate to bring more of the surface of a normal heart in contact with the chest wall than is usual. Such a condition can only be diagnosed through the signs of those morbid states of the lungs and pleura which might produce it, together with the absence of conditions which might produce cardiac disease, as well as other evidences of its presence.

CARDIAC DILATATION.

Dilatation of the heart is the most frequent cause of recognizable cardiac enlargement. As a physical evidence of that mechanical inability of the heart which marks dynamical failure of the heart muscle from all causes, it is of the utmost importance, both from a prognostic and a therapeutical standpoint. Its symptoms are a call for aid to the heart muscle, the urgency of which is in distinct proportion to the extent of the dilatation. As in practically all cardiopathies the occurrence of muscular inability is evidenced by the development of dilatation, the management of cardiopathies in general is practi-

cally embodied in the treatment of the various degrees of cardiac dilatation.

In relation to the heart itself cardiac dilatation is *primary* when it does not depend on previous disease of the heart muscle or valves; *secondary,* when valvular or mural disease results in dilatation. It may be *simple* (cavity increased, wall normal in thickness), *hypertrophic* (cavity increased, wall thicker than normal), or *atrophic* (cavity increased, wall thinner than normal). The latter form is infrequent but occurs most often in the auricles (right). The so called mixed form (Hayden) in which a portion of the wall is attenuated while other portions are thickened may constitute the earlier stages of aneurismal developments.

ÆTIOLOGY.—The general mechanical cause of cardiac dilatation is increase of intracardiac blood pressure. This may be an actual increase from obstruction to the blood flow or over-distention during diastole, or it may be a relative increase from loss of muscular power.

Simple dilatation, which is usually primary also, may occur from sudden and considerable increase in blood pressure from over-exertion, as in heart-strain from heavy lifting, running, bicycle racing or dancing. Those cases occurring from the hardships of war (Da Costa, Taylor, Fraentzel) and due to forced marches, badly adjusted accoutrements, etc., belong in this class, at least in some instances. Caley attributes cardiac dilatation to undue strain and myocardial weakness. To this we must add changes in the nervous mechanism of the heart.

The following are examples of simple primary dilatation:

Two young women aged 18 and 21 years, ballet dancers by occupation. Previous history good, present condition perfect with exception of heart. About ten days previous to presenting themselves they had undertaken an engagement which necessitated two lengthy and arduous performances daily. It was only by considerable effort that they were able to fulfill their duties. For last three or four days have suffered from dyspnœa and cardiac palpitation which obliged them to give up their performance. Examination showed that in both cases

the heart was arrhythmic under exertion. The apex was at
the left mammillary line in the fifth interspace. There was
distinct increase in the area of transverse dullness. Under
complete rest both cases recovered in about two weeks.

Boy aged 16. Previous history excellent. Made forty-
five miles on a bicycle over country roads in less than five
hours, the last fifteen miles being made at considerable speed
in order to keep up with an older and more experienced rider.
During last half hour of riding was very weak, could hardly
walk when he got off the wheel and was very short of breath.
Examination showed the pulse to be 120, remittent in force,
apex beat in left mammillary line. Complete recovery after a
few days of absolute rest.

In most of these cases it is the right ventricle which is
most affected because of the thinness of its walls, and, also,
because of the combined action of increased pressure both in
the systemic veins and in the pulmonary circuit. Thompson
calls attention to the effect of obstruction of the upper air
passages in producing dilatation of the heart, particularly of
the right ventricle. Simple dilatation may arise in such con-
stitutional conditions as anæmia, chlorosis, toxæmia, pneu-
monia, the eruptive fevers. Severe nervous strain or venereal
excesses may produce cardiac dilatation. Overdistention of
the heart cavities during diastole (as of the left ventricle in
aortic regurgitation) will cause simple dilatation, but this soon
passes into an eccentric hypertrophy through the development
of compensatory hypertrophy. Simple dilatation arises sec-
-ondarily to endo and pericarditis, toxic degenerations of the
heart muscle, myocarditis, the weakening of the muscle inci-
dent to Grave's disease or paroxysmal tachycardia. In all of
these conditions if the nutrition of the muscle is fair, the simple
dilatation passes into a condition of eccentric hypertrophy. If
the nutrition of the muscle is not sufficient to allow of perfect
compensation, then hypertrophic dilatation will occur.

Hypertrophic dilatation arises from all the obstructive
causes which result in hypertrophy. Whether the hypertrophy
be primarily simple or eccentric, as soon as degeneration of
the muscle develops, dilatation becomes the dominant feature

of the mechanical state of the heart. In all cases of hypertrophy with dilatation it is important to distinguish between primary dilatation with compensatory hypertrophy, and secondary dilatation from degeneration of an hypertrophied muscle. Hypertrophic dilatation develops from valvular insufficiencies, and in cases of chronic parenchymatous nephritis. In these cases hypertrophy and dilatation occur with such close coincidence that a stage of simple dilatation is not recognizable.

Sudden rise in blood pressure in the arteries may produce or increase dilatation in weak hearts. From this cause we may have dilatation of the left ventricle from spasmodic contraction of sclerosed arteries, or dilatation of the right ventricle from severe asthmatic attacks with a weak right heart. Skiagraphy has demonstrated the facility with which the heart dilates in accommodation for sudden and marked rise in blood pressure.

MORBID ANATOMY.—Dilatation of the heart may be general or partial, one or all of the cavities being affected. Considerable dilatation of one cavity will affect, to a greater or less extent, the other cavities. In general dilatation of the heart the organ will become rounded in shape and its breadth will be increased. If the right ventricle is affected the breadth of the heart will be increased, while if the left ventricle is affected the length of the heart will also be increased, though this depends largely on the presence of hypertrophy of the wall. The increase in the transverse diameter is the most noticeable feature on physical examination. The auricles are most frequently dilated, and the right ventricle is more often affected than the left. In mitral stenosis and in congenital lesion of the tricuspid opening great dilatation of the auricles may occur. Considerable hypertrophic dilatation of the right auricle will develop in congenital tricuspid stenosis. In aortic regurgitation very extensive hypertrophic dilatation of the left ventricle may develop. The compensatory hypertrophy of vascular sclerosis or interstitial nephritis will eventually pass into a condition of hypertrophic dilatation, or, if compensation is not developed quickly enough, dilatation and hypertrophy develop coincidently. The toxic degeneration of the heart muscle which may accompany parenchymatous nephritis will

induce extensive hypertrophic dilatation in a few weeks' time.

When dilatation results from loss of muscle power the tissue changes in the heart muscle will be identical with those of the morbid process in the heart muscle which has induced the muscular weakness. Thus they include all the varieties of inflammation and degeneration of the heart muscle. Where increased peripheral resistance has caused dilatation no changes may be demonstrable in the heart muscle. In some of the infectious diseases, as diphtheria, dilatation of the heart may be accompanied by degeneration of the muscle (Mosler), or perhaps by acute myocarditis (Leyden). In many cases of typhoid, typhus, erysipelas or pneumonia, muscular changes cannot be demonstrated though dilatation may have occurred. Here dilatation results from toxæmia of the muscle before pathological changes have reached a recognizable stage. In dilatation of the heart occurring with exophthalmic goitre, derangement of the cardiac innervation, venereal excesses late in life, or the excessive use of tobacco, definite myocardial changes resulting from these causes cannot usually be demonstrated. The exact relation of disturbed cardiac innervation to weakening of the cardiac muscle is not understood, but the clinical fact is indisputable.

With excessive dilatation of the cavities of the heart the trabeculæ are stretched and cord-like. With great ventricular dilatation the displacement of the papillary attachment of the chordæ tendineæ away from the center of the ventricular cavity prevents the closure of the valves of the auriculo-ventricular opening and causes regurgitation. Thus a mitral or tricuspid regurgitant murmur becomes indicative of the failure of hypertrophy of either ventricular wall to longer afford compensation. When dilatation reaches that degree in which the heart fails to empty itself on systole, we term the condition one of *asystolism*.

CLINICAL HISTORY.—The clinical history of cardiac dilatation comprises the clinical history of the hyposystolic period of all cardiac lesions. The most prominent symptom common to all varieties of dilatation is palpitation, which may be very distressing and may or may not be accompanied by præcordial distress or pain. There may be sighing respiration

and a sense of fluttering in the region of the heart. Dyspnœa
is complained of, more or less. Dyspnœa may occur only on
exertion or may appear in severe paroxysms. In right ven-
tricular dilatation the dyspnœa is constant and distressing,
being the chief complaint of the patient. Cough may be
troublesome, and when dilatation of the right ventricle results
from mitral regurgitation, the cough has a peculiar hoarse,
brassy, grating quality, characteristic of this condition.
Bloody expectoration may occur with dilatation of the right
ventricle. Vomiting and gastric distress are common in car-
diac dilatation. The extremities are cold, peripheral circula-
tion is bad, face pale, livid or cyanosed. Moderate jaundice
may occur. There may be partial suppression of the urine,
which may contain albumen or blood.

SYMPTOMS AND DIAGNOSIS.—In simple dilatation the ac-
tion of the heart is labored and more rapid than normal. The
rhythm of the pulse is not interrupted, but the radial pulse is
weak as compared with the character of the heart's action.
Moderate dyspnœa on exertion is present. In atrophic dilata-
tion and in advanced stages of hypertrophic dilatation the pulse
is rapid, feeble and irregular. The irregularity of the pulse
may simulate bradycardia in character, but on close observa-
tion it will be found that the slow radial pulse is due to ab-
sence of alternate beats at the wrist where the pulse may be
sixty while the heart beats one hundred or more per minute.
This kind of heart action is indicative of marked asystolism of
the left ventricle. The peculiar remittance in force of the
pulse which accompanies the gallop rhythm is often present.
The arteries are insufficiently supplied and the veins over-dis-
tended. In hypertrophic dilatation the heart action is labored,
tumultuous and may be very distressing to the patient. The
arterial trunks in the neck throb violently. The patient has an
anxious, distressed look.

In dilatation of the right ventricle dyspnœa is a promi-
nent symptom. If the dilation is from sudden strain of a
healthy right ventricle, the dyspnœa is sudden in advent and
panting in character, and is rapidly recovered from. In hyper-
trophic dilatation of the right ventricle, with degeneration of
the muscle, the dyspnœa appears slowly and is labored in char-

acter. It is generally preceded by fine, moist rales in the lowest portion of the lungs, usually most easily detected in the left lung. The dyspnœa may be so severe that the patient is obliged to sit up, and can hardly utter three consecutive words without pausing for breath. In some cases, particularly those hypertrophic dilatations associated with chronic nephritis, the respiration will develop a typical Cheyne-Stokes type. Typical Cheyne-Stokes respiration is a rhythmical alteration in the respiration in which there is a respiratory cycle of three periods: An apnœal period lasting from 30 to 45 seconds, during which no attempt at respiration can be detected; an ascending respiratory period lasting about 45 seconds, during which there is at first a scarcely visible effort at respiration which gradually becomes stronger until the breathing is deep and labored. During this ascending respiratory act the *inspiration* is forcible. A descending respiratory period lasting about 45 seconds, during which the breathing gradually grows weaker until this period merges into that of the apnœal. During the descending respiratory period the *expiration* is forcible. While Cheyne-Stokes breathing in cardiopathies is of serious prognostic import it is not necessarily an evidence of fatality.

In cardiac dilatation, inspection will show an increased area of cardiac impulse which may be indistinct or forcible. The apex impulse is not well defined and is more or less difficult to locate. Epigastric motion will be present in dilatation of the right ventricle.

Palpation determines the heaving, undulating character of the cardiac impulse, the displacement of the apex beat toward the left and the indefinite character of the apex impulse. In simple dilatation the apex will be displaced to the left, but will remain on the same horizontal plane. In hypertrophic dilatation the apex will be displaced to the left and downward, but the relative amount of displacement to the left as compared with the downward displacement, determines, to an extent, the various degrees of dilatation and hypertrophy present.

On percussion we obtain an enlarged area of cardiac dullness. In general dilatation the area of dullness will be somewhat oval in shape. In dilatation of the right side, dullness

may extend to the right nipple. When the left side is affected, dullness may extend to the left axillary space. Dilatation of the auricles extends the dullness upwards, perhaps as far as the first rib. By some, the results of percussion are not deemed essential to the diagnosis of cardiac dilatation (Musser). They are frequently not necessary, but can hardly be styled unessential, as many of the other symptoms may be present in degenerated hearts without dilatation. When consolidation or sclerosis of lung tissue, or intrathoracic tumors occur in the cardiac region, the direction and extent of the cardiac dullness may be very important.

Auscultation shows the heart sounds to be feeble and indistinct. The first sound is weak, muffled or altogether lost. The second sound may be the loudest all over the præcordial area or may be inaudible at the apex, while over the aortic or pulmonary valves it is much the louder of the two heart sounds. The advent of right ventricular dilatation in pneumonia will be indicated by a short, quick, high-pitched pulmonic second sound. At times a gallop rhythm may be heard; by some it is given as a sign of cardiac dilatation, but in my experience it is rarely heard in its true form in dilatation of the heart. [With a struggling, irregular heart, where only every other contraction produces a radial impulse, and a pulsus bigeminus, the first cardiac sound of the second or weaker contraction of the heart, may be quite inaudible. This gives three appreciable heart sounds for the two contractions of the heart, and as the second sound of the heart is louder than the first, the second of the three sounds is the loudest. The difference between this triple rhythm and that of a true gallop rhythm, is that in the latter case the long interval is between the first sound and the second or loudest sound; while in the former case the long interval is between the second or loudest sound and the last sound, or second sound of the second weaker contraction.]

With considerable dilatation of either ventricle a systolic murmur may develop at either the mitral or tricuspid opening. If organic murmurs have been present they will become changed in character or will disappear entirely. With considerable dilatation of the right ventricle œdematous rales will

appear at the lowest portion of the lungs. A pulmonic regurgitant murmur may attend extensive dilatation of the right ventricle.

The most important symptoms in the diagnosis of cardiac dilatation are increased area of dullness, displacement of the apex beat, pulsus bigeminus (indicates asystolism), soft systolic murmur in mitral or tricuspid area, dyspnœa, distention or pulsation of jugular veins (right ventricular dilatation), congestions, œdema and cyanosis.

TREATMENT.—In the treatment of cardiac dilatation is included all the measures which may be applied to failing heart. Whatever means are adopted, no routine methods of application are admissible. The individual case is a law unto itself, and the relation of capacity and ability to resistance, must be carefully estimated in each instance. The indications for treatment in simple dilatation from heart-strain or acute disease, will differ materially from those in hypertrophic dilatation from increased resistance.

The recognized methods of treatment for inability of the heart are *rest, diet, medication* and *exercise.* Rest is absolutely necessary in all cases where a normal or temporarily weak heart muscle has given way to strain which has no permanent element of increased resistance, and where we desire to restore normal conditions and not to develop additional power. Thus rest applies to simple dilatation from heart-strain, infectious disease, or acute myocarditis; rest is also necessary in advanced hypertrophic dilatation where even temporary rise in blood pressure is dangerous. Dyspnœa, cough, palpitation, cyanosis and dropsy all yield more readily to other methods of treatment when rest is insisted upon. The judicious combination of rest with the milder forms of mechanical therapeutics affords the most effective mode of treatment in marked failure of the heart which threatens to develop asystolism.

Diet is very important in cardiac dilatation. The general tendency is toward over-feeding. Many cases do best when placed on a restricted amount of food. The diet should be nourishing and easily assimilated and should be governed by the same rules as in cardiac degeneration. A routine diet

list is not advisable except in cases of dilatation due to over-feeding and drinking. The use of liquors should be restricted to small amounts of light dry wines at meal times. Whisky and brandy should be prohibited. As temporary stimulants they may give relief, but their continued use, even in small doses, is detrimental.

The medicinal treatment of cardiac dilatation will not be considered here further than to say that it is not, as yet, displaced by any other method of treatment, and when properly combined with rest and diet, constitutes our most effective means of overcoming the advance of cardiac failure.

In all ataxic conditions of the heart we aim at restoration of the normal relation between the dynamic power of the heart and the resistance against which the heart has to work, or, at least, to modify abnormalities of this relation as much as possible. In order to accomplish this, one must increase the dynamic power of the heart when it is lost, and lessen the peripheral resistance when it is increased. One of the most effective means of accomplishing both of these objects is through the therapeutic application of exercise. Long ago, Ling demonstrated the therapeutic utility of massage in cardiopathies, and Oertel has elaborated the uses and value of walking and mountain climbing in diseases of the heart. Both of these systems are in accordance with the teachings of Stokes in regard to the management of chronic cardiopathies.

Of late years, attention has been directed chiefly toward the Schott method of resistance exercise (*Winderstandsgymnastik*) combined with medicated baths. The utility of the Schott method cannot be disputed. Considerable discussion has arisen as to its mode of action, and its therapeutic application and limitations.

The observations of Ludwig, Sadler, Mosso, Gaskell, Chauveau, Kaufmann, Marey, Oertel, von Basch, Bloch, Brunton and Tunnicliffe, and others, while differing somewhat in specific results, because of necessary variations in the grade of exercise and the circumstances under which experiments were made, agree in the main as to these facts: Exercise promotes the flow of blood through the muscles, from the arterioles into the veins; there is, during exercise, a primary

and temporary rise in blood pressure, followed by a fall in pressure which is of greater or less duration. This fall in pressure is an important feature of the effect of exercise and baths as far as therapeutical application is concerned, for we will find that the class of cases most benefited by these measures are those in which the heart is more or less constantly opposed by unusual resistance. The fact that baths and exercise may be contra-indicated in advanced arterial sclerosis does not invalidate this statement, for in such cases it is not the fact of hypertension which contra-indicates the treatment, but the dynamic insufficiency of the heart muscle which is unable to withstand the temporary increase in pressure incident to the employment of mechanical methods of treatment. The capacity of the peripheral circulation, as demonstrated by the experiments of Ludwig, offers further reason for the efficiency of mechanical therapeutics.

The slowing of the pulse which is observed during and after the use of baths and exercise in suitable cases, notwithstanding the lowering in blood pressure, has been explained through a probable stimulation of the pneumogastric (Broadbent and others).

The remarkable diminution in the size of the heart which it is claimed is produced by baths and exercise, is not unreservedly accepted by all observers. Vivian Poore raised the question of the effect of increased pulmonary expansion in modifying the area of cardiac dullness. Bezly Thorne, one of the most enthusiastic advocates of mechanical therapeutics, has produced some elaborate theoretical explanations for those physical alterations in the heart, which he claims can be demonstrated by auscultatory percussion. Some of the best clinicians are not as optimistic as Thorne in regard to the possibilities of auscultatory percussion. Broadbent says that Thorne's tracings demonstrate the fallacy of this method of examination. Herringham believes it not superior to mediate percussion. Little does not believe the cardiac outlines can be so accurately determined during life as to permit of such definite conclusions from percussion.

My own observation has been that after exercise, and especially after the bath, there is an increase of from one inch

to one and one-half inches in chest expansion; that there is
from one-quarter to one-half inch diminution in the transverse
diameter of cardiac dullness with a corresponding displace-
ment upward and inward of the apex beat. I have observed
these changes in cases in which the heart was not favorably
affected by the bath, and in which the dynamic increase in the
power of the heart muscle was exceedingly transient. Un-
doubtedly there is diminution in the size of a dilated heart as
a result of mechanical therapeutics properly applied in suitable
cases, but the observation of Poore is very pertinent to the
effect that the amount of change recorded in many cases is not
consistent with the assumption that it is entirely due to shrink-
age of the heart itself. Röentgen radiography has demon-
strated that changes in position of the heart are responsible for
some of the remarkable results of percussion.

Marked conditions of arterial sclerosis or nephritis are
admitted contra-indications to the use of baths and resistance
exercises (Osler, Camack, Brunton, etc.), though Bezly
Thorne advocates their use in vascular sclerosis and even in
some cases of aneurism. According to Tyson, the typical con-
dition for mechanical therapeutics is a weak, dilated heart;
deranged circulation with frequent, feeble, irregular pulse;
dyspnœa, engorgement of venous circulation and of the liver
and kidneys with scanty urine. If these symptoms always
represented a definite degree of dynamic failure of the heart
muscle such a generalization might be proper. These symp-
toms, however, may be the only manifestations of an actually
present or impending asystolism, and are therefore obviously
unsafe as a guide for the applicability of mechanical thera-
peutics. Osler thinks the Schott method applicable to dilated
hearts where there is not much muscular degeneration; in
young subjects, or from 40 to 45 years of age, in whom there
has been excessive consumption of food and drink. Brunton,
from his experiments, concludes that where the heart is too
weak to bear the primary rise in blood pressure caused by even
gentle exercise, massage should be used; when the heart is
strong enough to bear the rise in pressure, exercise is best as
the resulting fall in blood pressure is greater in amount and
of longer duration. Grainger Stewart says that with grave

debility of the heart, use only passive exercise; with stronger muscle use Schott exercises; with sufficient cardiac tone, use Oertel method of hill climbing. The baths may be tried in all cases except those suited only for passive exercise.

It is evident that the application of mechanical therapeutics to cardiopathies necessitates an intelligent individualization. That these methods have potentialities of great harmfulness cannot be denied by any one who has given them clinical exemplification. It is surprising, at first, how little resistance it takes on the part of the operator to cause dyspnœa in a patient when giving resistance exercises.

We may grade our measures of exercise into three classses: 1. *Passive exercises*. These exercises were originated by Ling. The movements are given by the operator, with entire passiveness on the part of the patient. They consist of rolling and kneading of the foot, kneading of the calf and thigh, movements of the arm, flexion of knees and rotation of hips; then hands and arms; massage of abdomen, percussion of præcordial area and intercostal spaces of left side, manipulation of spine, arm movements to expand thorax. 2. *Movements with limited resistance* (Schott). These constitute a modification of the Swedish system, and consist of a series (nineteen in number) of flexions, extensions, adductions and abductions of forearm, leg, thigh and trunk. Each motion is made against slight resistance on the part of the operator, and each act of flexion or extension is followed by a short rest. 3. *Climbing method* (Oertel). This method has a more restricted application, and is adapted to those cases which have not reached, or have improved beyond, the conditions necessitating the first two methods.

In giving exercise treatment, great judgment is required on the part of the operator. The treatment should be suspended on the first evidence of distress, as indicated by dyspnœa (shown by inspiratory dilation of alæ nasi), palpitation, or irregular pulse. The good effect of exercise is indicated by slower pulse, diminution in area of cardiac dullness, easier respiration, greater ease and comfort of patient.

The baths may be used in all cases not limited to the first grade of exercise. Whether their good effects are due to

stimulation of the peripheral nerve endings by the carbonic acid gas is a question; the carbonated baths however, are more powerful for good than the plain medicated bath. The artificial bath can be given at home or in a hospital without much difficulty. The baths should be given in the morning on an empty stomach. The patient should make no unnecessary exertion. A forty-gallon bath should be used, and the entire body should be immersed. Light friction of the body may be used while in the bath, but it is not necessary. The bath should not be prolonged over eight or ten minutes. Any unusual irregularity of the pulse, cyanosis or apnœa, while the patient is in the bath, indicates removal at once. After removal the patient should be well rubbed down, placed in bed and kept absolutely quiet for an hour.

Plain medicated baths may be used at first in advanced cases. This bath consists of four or five pounds of sodium chloride and six or eight ounces of calcium chloride. The carbonated baths should range in strength from one containing sodium chloride six pounds, calcium chloride ten ounces, bicarbonate of soda six ounces, and hydrochloric acid (twenty-five per cent.) eight ounces; to one containing sodium chloride eleven pounds, calcium chloride twelve ounces, bicarbonate of soda one pound, and hydrochloric acid (twenty-five per cent.) two pounds. In carbonating the bath the bottle containing the acid may be inverted below the surface of the water, the stopper removed and the acid diffused through the bath. It is not desirable to put all the acid in at once as it will escape too quickly. When convenient the gas may be introduced from a cylinder of compressed gas.

The Oertel method of hill climbing is adapted to patients whose condition is largely due to over-feeding. For these patients great possibilities exist in the Oertel system of exercise.

During the last five years a combined means of pleasure and exercise has developed to such an extent in the bicycle, that physicians are constantly obliged to decide for or against the use of the wheel in cardiopathies. The one great danger of the bicycle as a health exercise is the ease with which the exercise can be carried to excess, and this very facility of execution constitutes at once the pleasure and benefit of wheel

riding. There are few forms of exercise which affect so many muscles of the body and at the same time tax the respiration so little as wheel riding under the proper conditions. Barring the danger of overdoing, and keeping certain facts in mind, there is no reason why the wheel should not be used with advantage in many organic or functional diseases of the heart. Persons exhibiting subjective signs of cardiac failure, should not, of course, ride a wheel. It is therefore limited to cases of functional disease or fairly well compensated lesions. Heart patients who ride wheels should bear in mind these facts: do not ride over six miles an hour, confine riding to a boulevard or smooth gravel road if possible, do not ride steep grades, do not ride against a strong wind, adjust saddle well forward so that the weight of the body can be utilized in propelling the wheel, raise saddle so rider's leg is almost straight when pedal is low, use upturned, wide handle bars.

I have known heart patients to derive great benefit from wheel exercise and do not hesitate to allow its use under proper restrictions, except in cases of mitral stenosis and marked aortic regurgitation, in which conditions, even though the heart muscle be good, wheel riding should not be allowed.

CHAPTER VII.

CARDIAC NEUROSES.

Under the title of neuroses we wish to consider those abnormalities of cardiac function and rhythm which are symptomatic of the various modifications of cardiac innervation, nutrition and metabolism that, while present to some extent in all cardiopathies, yet find most pronounced symptomatic expression, at least so far as the perversion of innervation is concerned, in the circulatory conditions which attend maturer years. Balfour has grouped these conditions under the general subject of "The Senile Heart," and the severity of the symptoms in relation to the difficulty of demonstrating any definite cardiac lesion, justify their being regarded in many instances as manifestations of cardiac senility, for, when present in young individuals, these symptoms can frequently be connected with some definite cardiac lesion. The only conditions which are invariably present in this class of cardiopathies are imperfect nutrition of the heart muscle and disturbance of cardiac innervation. No definite pathological state can as yet be held responsible for the cardiac neuroses, though they may be manifest in any of the various pathological conditions in which the factors mentioned obtain.

[If we recall a few facts relative to the innervation, circulation and action of the cardiac muscle, we will more readily appreciate the complex possibilities for derangement of function, with or without physical alterations of a demonstrable nature.

The basic cardiac plexus of nerves is connected with the cardiac ganglia. These ganglia are scattered through the cardiac substance, particularly at the junction of the sinus venosus with the auricle, and in the auriculo-ventricular sulcus. —His and Romberg have shown that the ganglion cells of the heart have the same origin as all other sympathetic cells, and

suggest that these ganglia form impulses which regulate the circulation through the vagus and accelerator nerves. The rhythmic nature of the contractions of the heart has been attributed to these ganglia, but at present it is regarded as an inherent automatic property of the heart muscle, myogenic in nature (the power of spontaneous movement possessed by all primordial protoplasm—Foster), and independent of the nerve ganglia (Botazzi). Kronecker claims to have demonstrated a co-ordination center located at the junction of the upper and middle third of the ventricular septum. The location of this center (in the area supplied by the anterior coronary artery) has some clinical interest. The ultimate distribution of the cardiac nerves is in great profusion through the heart muscle, underneath the endo and pericardium, and about the blood vessels. Berkeley has demonstrated their termination between and on the muscle fibres. Smirnow claims to have demonstrated sensory nerve endings in the cardiac connective tissue, which he thinks are connected with the depressor nerve.

The basic plexus unites into three cords the superior and inferior cardiac nerves (branches of the vagus), and the accelerator nerve (sympathetic). The superior and inferior nerves, usually called branches of the vagus, have separate origin and function, and the inferior nerve is more closely associated with the spinal accessory than with the vagus nerve. The superior cardiac nerve is an afferent nerve and conveys influences to the vaso-motor center in the medulla which modify and regulate the pressure in the arterioles. The action of the inferior cardiac nerve is *anabolic:* it inhibits the action of the augmentor nerve, it slows and reduces the force of the heart. Its action is chiefly on the auricles whose action it may entirely arrest. The excitability of the ventricles is diminished and their output may be reduced thirty per cent. (Roy and Adami) Their action may be temporarily arrested by strong stimulation of the vagus, or, under certain degrees of stimulation, they may develop action entirely independent of the sinus and auricles, which is frequently the cause of irregularity of the heart.

The action of the accelerator nerve is to increase the force and frequency of the cardiac contractions (von Bezold). These effects are not necessarily coincident, sometimes one

and again the other being most prominent (Foster). The accelerator nerve is the *katabolic* nerve of the heart; it is through it that cardiac metabolism is effected and energy produced (Gaskell).

The arterial supply of the heart is remarkably ample in comparison to the size of the organ. The coronary arteries are subject to both diastolic and systolic pressure. The left coronary artery is the largest, is most frequently involved in sclerotic, atheromatous or syphilitic processes, and, from its distribution, is the most important in connection with morbid processes of the cardiac muscle. The anterior branch of the left coronary artery runs in the anterior inter-ventricular groove and supplies the anterior wall of the left ventricle and the septum. It is most frequently the seat of arterial disease and of thrombosis or embolism. Osler has called it the artery of sudden death. The effect on the heart of interference with the coronary circulation is in direct proportion to the size of the artery involved. Porter's experiments show that ligation of the smallest artery (arteria septi) does not arrest the heart's action; of the next largest (coronaria dextra), arrest in fourteen per cent.; of the next largest (descendens), twenty-eight per cent.; and of the largest artery (circumflex) sixty-four per cent. Porter also shows that ligation of a coronary artery is followed by a rise in auricular pressure which may possibly interrupt the entire coronary circulation. Although the coronary arteries are not, strictly speaking, terminal arteries, the anastomosis is not free enough to allow of the establishment of collateral circulation. The effect of plugging of an artery is to produce an anæmic infarct. These occur most often in the walls of the left ventricle and in the septum near the apex. They may be firm, white or opaque in color, and may have a prominent outline. They are gradually changed into fibroid patches.

The effect on the heart's action of plugging of a coronary artery is to produce fibrillary contraction with loss of co-ordination which is difficult to re-establish. It is evident that narrowing of the orifice of the coronary arteries may be quite as serious a matter, or, indeed, even more so, than extensive disease of the arterial walls.]

PRÆCORDIAL PAIN.—Among the most indefinite and troublesome neurotic states of the heart are those accompanied by præcordial pain. Pain about the heart is a condition not necessarily indicative of, or invariably present in, any morbid state of the heart. Præcordial pain may occur from flatulent dyspepsia, gastralgia, intercostal neuralgia, myalgia, pleurisy, pericarditis, anæmia, neuro-vascular spasm of central origin (angina pectoris vasomotoria—Nothnagel), peripheral vascular disease, aneurism or tumor at the base of the heart pressing on the basic plexus, disease of the coronary arteries or heart muscle (idiopathic angina pectoris), aortic valvular disease, atheroma or distention of the aorta, distention of the heart cavities in mitral disease or in acute simple dilatation of the heart. Heart pain is too inconstant to be regarded as belonging to the symptomatology of organic heart disease. A most serious affection may be painless while another of no special import may exhibit severe pain. Cardiac pain may vary from mere præcordial anxiety or consciousness of heart's action with the peculiar indescribable sensation associated with palpitation, through intermediate states of tension, constriction, grasping, boring, sticking, dull, heavy or piercing pain which may be constant or paroxysmal. The toxic form of præcordial anxiety, pain and distress which results from the use of tobacco is largely due to the gastric irritability induced by the weed, and in the treatment of these cases we must insist on stopping the use of tobacco. The general treatment of præcordial pain is symptomatic and includes tonic, dietetic and hygienic management.

ANGINA PECTORIS.—This is the most important, as well as the most sudden and alarming, condition accompanied by heart pain with which we meet. Characterized by no definite pathology other than changes in the heart muscle, nerves, and arteries which may occur without anginal manifestations, it may yet be classed, on a symptomatic basis, as a neurosis.

True angina pectoris may be classed as a part of the symptom complex—a syndrome (Osler)—of those varied lesions of the coronary nervous system, arteries and muscle which result in ischæmia of the heart muscle with depression of its force and energy (Balfour). The recognition of a spurious

type of angina pectoris (*angina pectoris notha*—Latham) is regarded by Balfour as "a mark of ignorance rather than a refinement of diagnosis." Osler, while not commenting on the scientific relations of such a classification, says that it has "practical advantages which far outweigh any theoretical objection." In consideration of the difficulty of identifying the two cardinal symptoms of true angina (*dolor pectoris et angor animi*) in some instances, and also, the occurrence of true angina without pain (*angina sine dolore*—Gairdner), we must admit the force of the latter's position. Under the designation of pseudo-angina would come those cases described by Walshe, occurring in women and due to hysteria, neuralgia and spinal irritation; the various toxic anginas, and the vaso-motor forms identified by Nothnagel.

Angina pectoris vera usually occurs after the age of fifty (eighty per cent. of all cases occur after fifty—Quain). It is more frequent among the educated, well-to-do classes and is seldom met with in hospital practice. Professional men frequently develop angina pectoris vera. It is more common in men than in women (42 in 227 cases—Huchard). Some hereditary tendency toward angina pectoris is shown in families having a tendency to arterial degeneration, and it may be associated with gout, syphilis, diabetes, the weakened heart of post-febrile states, locomotor ataxia (Leyden), and emotional conditions may cause true (?) angina pectoris. While the ætiology of the disease is not identical with arterio-sclerosis it is so closely connected with degeneration of the coronary arteries that the true paroxysm is seldom found apart from that condition (Osler).

The clinical history of angina pectoris is variable. The paroxysms may recur every day or so, and within a few days or weeks develop a condition of asystolism (*l'etat de mal angineux*—Huchard); they may extend over a period of months or possibly years (the most common form). The first well marked paroxysm may be at once fatal, or sudden death may occur soon after the occurrence of a paroxysm and without any further manifestation of angina, as occurred in the following case:

Mr. L., aged sixty-one years; business man. Previous history good. General condition excellent with the exception of slight arterial sclerosis, though this was not more marked than is frequently found in men of his age. He had recently had some family and business troubles which had worried but apparently not depressed him very much. I was called to his place of business about five o'clock, P. M., and found him very pale, with an anxious expression of countenance. He said he had a severe pain in his heart, "a peculiar pain, as if the heart was being squeezed to a pulp by some great force." The pulse was regular but rather weak. He was taken home in a carriage and given nitro-glycerine and morphine. By half past six o'clock he was quite comfortable. I called at eight o'clock and was told that he had just gone upstairs to his bed room. I followed him in about ten minutes and found him sitting in a chair, dead. He had but the one attack of pain. While the pain was severe he said he did not mind it so much as he did the awful sense of dying, which he seemed surprised at being unable to control, "for," said he, "I am not afraid."

An attack of angina pectoris may be precipitated by the slightest exertion in the recurrent form of the disease. Mental emotion, the effect of cold on the surface of the body, digestive disturbances, especially gastric or intestinal flatulence, may cause a paroxysm. Again, it may occur without any exciting cause being apparent. The chief symptoms of the attack are pain (*dolor pectoris*) and the mental anguish (*angor animi*). The pain may be of any degree of intensity and of varying character. It may be the chief symptom or may be subsidiary to the sense of torture. Again, there may be little or no actual pain (*angina sine dolore*—Gairdner). The pain may be in any part of the præcordial area, substernal (sternalgia), or in the scrobiculus cordis. It begins suddenly and may end abruptly. The radiation of the pain is in the first to the fourth dorsal areas, or even to the ninth dorsal area (Head). It usually extends to the left shoulder and arm, though it may extend to the sides or back of the chest, right shoulder and arm or remain stationary. Areas of cutaneous hyperæsthesia may be present about the chest (Mackenzie). Numbness, paralysis or

spasm of the muscles of the left arm may follow an attack.
The muscles in the hand which are suplied by the ulnar nerve
may atrophy (Eichorst). The pain of angina has been attrib-
uted to cramp of the heart muscle; pressure in, and stretching
of the heart cavities; arterial pain; neuralgia. Musser says the
pain is due in part to increased intraventricular pressure and
may disappear when dilatation supervenes. Balfour thinks the
pain is accounted for by relative or actual ischæmia of the
heart muscle, at times in connection with vaso-motor disturb-
ances and lowered nerve function.

The mental anguish is often the most distressing symp-
tom. It is indescribable and is generally present in severe
cases. During the attack, the patient is unable or unwilling to
move, the skin has a deadly, ashy pallor, is bathed in cold per-
spiration or may be, at first, deeply suffused or congested. The
pulse is weak and feeble but is usually regular during the at-
tack, though it may be changed in rate and rhythm if myocar-
dial disease be present. The respiration may be natural but
usually is shallow, jerky, or may be suspended at times during
the severe pain of an acute attack. Cheyne-Stokes respiration
may be present between the attacks. Bronchial symptoms may
accompany or follow an attack. Cardiac asthma may be pres-
ent with an attack of angina and in vaso-motor forms of angina
sine dolore may be regarded as symptomatic of the latter state.
Dyspnœa is not a feature of true angina (Heberden), yet if
the heart muscle be incompetent, any degree of dyspnœa may
be present. Gastro-intestinal symptoms may be the most pro-
nounced. Nausea, vomiting, flatulency, hiccough or gastral-
gia may be well marked, as in the following case:

Mr. B., aged sixty-one years; business man. Was
troubled for several years with dyspepsia and was exceedingly
careful about his diet, as the slightest indiscretion would bring
on an attack of pain in the region of the stomach. During the
last year has had three attacks of very severe pain which came
on suddenly and ceased abruptly, and was followed by marked
depression. The pain did not radiate although there was some
substernal pain. He was positive as to its location in the stom-
ach. The mental state during the attack did not seem to be

prominent. He regarded his trouble as gastric in origin, though his physician assured me that the pain was undoubtedly anginal in nature. No evidence of physical change in the heart could be detected. Three months after my examination he had a severe attack in the evening on retiring and died in half an hour. There was a moderate degree of coronary sclerosis, also of interstitial myocarditis.

There are many cases which do not manifest the cardinal symptoms of a typical attack of angina, and among these cases which do not present the more characteristic symptoms we will find certain more or less well marked types, such as *syncope amiginosa* (Parry), in which there is faintness or temporary loss of consciousness; the *Adams-Stokes syndrome,* characterized by vertigo and syncope, with slow pulse (may be as low as 5-10 per minute—Osler), and apoplectiform attacks which may resemble syncope anginosa; *angina pectoris sine dolore* (Gairdnér), in which pain is subsidiary to subjective sensations of distress (angor); and an *asthmatic* type in which cardiac asthma is the principal feature. (Cardiac asthma is a vaso-motor angina sine dolore—Balfour.) The terms cardiac asthma and cardiac dyspnœa are now much used synonymously. The type of breathing in actual failure of the heart muscle (dyspnœa) is different from that in sudden relative incompetence of the heart muscle from increased peripheral resistance (asthma) due to contraction of the arterioles. The latter is neurotic in nature, of sudden onset, and nocturnal in advent. The actual *air hunger* in either case is due to the same cause—diminution of the alveolar capacity from dilatation of the pulmonary vessels and diminished elasticity of the lungs (von Basch).

The following interesting case, seen with Dr. F. C. Wells of this city, may be classed as syncope anginosa. I saw the patient on the evening of March 1st about an hour after his second attack of the evening. Dr. Wells, who had observed him closely during several of these attacks, furnished the following information:

Mr. M., business man aged 55. For three years has had peculiar feelings in his head, would seem to lose himself for a few minutes. These symptoms would occur at night at intervals of five or six months. For ten or twelve years has had headaches in back of head and frontal region. These headaches recur at intervals between the attacks of partial unconsciousness. In Nov. '96, had a severe attack of syncope. Dr. Wells saw him after attack had subsided. His pulse tension was high, the pulse was regular but slow. About an hour after he had another attack, less severe and with but partial unconsciousness, after which the pulse was weak. On March 1st, '98, had a severe attack at seven o'clock in the evening while eating dinner (previous seizures had all occurred late at night), he fell to the floor unconscious but recovered in a few minutes. This was followed by another severe attack at eight o'clock. The patient realizes for an instant that attack is coming on. In the severe attacks the pulse stops suddenly and cannot be felt at the wrist, nor can the heart's action be felt or heard. Unconsciousness occurs at once with cessation of respiration. The pulse gradually returns in from fifteen to twenty-five seconds. Respiration gradually returns and is wheezing in character with bronchial rales. During the attack the surface of the body is very pale and bathed in cold perspiration. The principal factor apparent in producing an attack was the nervous state incident to business strain and physical exhaustion. Direct physical exertion does not seem to produce the paroxysms.

PSEUDO-ANGINA PECTORIS.—Without debating the abstract propriety of recognizing a false angina we may note that Walsh's classification is virtually adhered to by many. We observe many cases of cardiac pain in which the pain is the only symptom in which they resemble true angina. Morison says that as far as angina is not associated with a demonstrable lesion and tends to get well, we may recognize a spurious angina. If we extend this idea somewhat it is no wonder that angina pectoris was regarded by some of the writers (Læennec) as so very common. According to Huchard the pseudo-anginas are due to neuralgia of the cardiac plexus from vaso-motor neurosis or other causes. The chief distinguish-

ing features of false angina are the inability to demonstrate, or to reasonably infer, the presence of cardiac or coronary disease; the absence of the element of angor animi from the attack; the presence of various ætiological factors in the attack, of reflex origin; its more frequent occurrence in women and in early life, and its non-fatality. False angina pectoris is usually of neurotic or toxic origin. The neurotic form occurs in hysterical or neurasthenic individuals, from the various reflex visceral irritations, or from vaso-motor disturbances. [Vaso-motor disturbances are so common in all forms of angina pectoris that it is difficult to tell whether the vascular state has an actual or relative connection with the angina. The type described by Nothnagel, with primary vaso-constriction and secondary embarrassment of the heart, in which a general angeiospasm throws great strain on the left ventricle, is frequently met with. Douglas Powell claims that these cases constitute a lage proportion of the cases of angina pectoris. The relation of vascular spasm to all forms of angina is an interesting question; whether the vaso-motor symptoms of true angina are due to afferent impulses from the heart, or whether these vascular disturbances, as the primary factor in the production of an attack, add the last straw to a weak, improperly nourished heart, are debatable questions. The possibility of local coronary angeiospasm, must be considered in toxic and reflex cases.]

The toxic anginas occur from the abuse of tea and coffee, tobacco and from the various toxæmias. These causes, while usually resulting in disturbances of cardiac rhythm, may produce severe angina. The rapid heart, irregularities, palpitations, uneasiness in the præcordium, darting pains in the cardiac region or sense of oppression from tobacco dyspepsia, are all common symptoms in those who use tobacco excessively. Distinct anginal attacks from tobacco are rare. Huchard describes three forms: A benign form due to tobacco dyspepsia; a functional form due to coronary spasm; and an organic form due to organic contraction of the coronary arteries from nicotine arterio-sclerosis. Balfour says that tea, tobacco, etc., cause angina by enfeebling the heart and causing relative ischæmia.

The following case was one of pseudo-angina caused prin-
cipally by tobacco:

Dr. N., aged 59 years, of full habit, always healthy but
troubled with fermentative dyspepsia, particularly if he used
tobacco freely. He had never been a very heavy smoker, and
did not chew, but tobacco affected him badly. For last two
years noticed distress about his heart if he smoked freely.
During the last year has had four attacks of rather severe car-
diac pain coming on in the evening after smoking. One attack,
after a day of severe nervous strain, came on immediately as
he began smoking. I saw him during his fifth attack. His
face was slightly pale, pulse regular but feeble and of high
tension. The pain was quite severe and radiated to the left
shoulder and left side of the neck. The respiration was not
affected. A morphine injection relieved the pain and brought
the pulse up full and round. The patient is quite satisfied as
to the connection between the tobacco and the angina.

The therapeutic management of angina pectoris is not as
easy to formulate as it would be were our ætiological and
pathological classifications of the disease more clearly defined.
Douglas Powell says that "these difficulties diminish when we
recognize that there is a continuity in the phenomena presented
to us for treatment from the slightest degree of introspective
recognition of the cardio-vascular mechanism, through the
more distinct evidences of cardiac anxiety and distress in con-
nection with the higher and more persistent grades of arterial
tension to the paroxysmal attacks of acute breast pang associ-
ated with a veritable asthma of the blood vessels which may
supervene in any case of the series; and this continuity is to
be observed between the so-called false and true angina."

In the treatment of a paroxysm of true angina if gastric
or intestinal distress has instituted the attack, as is very likely
to be the case, we may give a saline cathartic, or administer
carminatives if there is distention from gas, in which case
camphor, Hoffman's anodyne, capsicum, chlorodyne (liquid
or tablets), or in some cases bromide or bicarbonate of sodium
will be useful. For the relief of the pain, inhalations of chlo-

roform or nitrite of amyl may be used until the slower remedies can act. Nitroglycerine may be used in doses of from 1-100 to 1-50 of a grain, particularly if arterial sclerosis is marked. The best remedy for the pain is morphine hypodermically in one-quarter grain doses repeated in half an hour if necessary. (Bostwick reports a good result in an obstinate case, from hydrobromate of hyoscine, 1-75 of a grain given hypodermically,—a rather large dose ordinarily.) In rapidly repeated attacks it may be necessary to keep a patient partly under the influence of morphine for some time. Brunton recommends amyl nitrite and hot applications for the paroxysms of angina pectoris. Iodide of potassium (5 to 15 grains) is best to remove the cause. It should be continued for from ten to fourteen days.

In a man aged 63, with marked arterial disease, enlarged heart, marked arrhythmia, constant dyspnœa, severe spells of cardiac asthma and frequent attacks of cardiac pain (*l'etat de mal angineux*), finally culminating with Cheyne-Stokes respiration in asystolism and death, life was maintained and some measure of comfort afforded for several months, by continuous alternate dosing with ¼ grain of morphine and 1-100 grain of nitroglycerine every two to four hours.

Pawinski describes an exocardial angina associated with dry pericarditis, which is not amenable to treatment by the vaso-dilators and must be treated like pericarditis of rheumatic origin. It is characterized by pain at the middle or base of the sternum, epigastrium, or over the cardiac area. There is a to-and-fro friction sound of pericarditis.

The management of the patient with angina pectoris involves first of all surety of diagnosis, for, to modify or sacrifice one's business prospects for what may prove to be a functional difficulty of no great danger, will lay the physician open to criticism. Again, failure to properly regulate the patient's habits, may be fatal to him. This does not mean that every sufferer from angina pectoris must fold his hands and wait for death. There is no necessity for idleness, for it affords no better prospect, nor indeed, even as good, as moderate employment of body and mind. All mental worry and strain must be avoided. Exercise must be taken with limitations which

each patient must learn for himself. Diet is very important and the general rules of diet and hygiene which apply to the ataxic period of all cardiopathies are especially applicable here. Every patient should understand the effect on the vascular pressure and the heart, of mental and physical fatigue or of gastric disturbance in his particular case.

Medicinally, all systemic conditions as gout, rheumatism, syphilis, diabetes, etc., should receive attention. In most cases we will use those remedies which improve the tone and function of the arteries and heart, such as strychnia, arsenic and the iodides. The latter are particularly indicated when there is arterial sclerosis. They should be given for long periods in moderate doses (ten grains of potassium iodide, or five to eight of the iodide of sodium, thrice daily, more or less continuously for years). Full doses of strychnia (1-20—1-30 grain) should be given more or less continuously. When arterio-sclerosis is marked, continued treatment with nitroglycerine (1-200 to 1-100 grain) or nitrite of sodium (2 to 5 grains) may be advisable. For the syncope of anginal attacks we may use aromatic spirits of ammonia, brandy, whisky, nitroglycerine, ether or camphor hypodermically. In some cases when the heart acts badly after consciousness has returned, digitalis is useful. In heart failure from angina, which does not respond to ordinary methods, cardiocentesis may be employed to revive the heart (Osler). In pseudo-angina pectoris the treatment is symptomatic as far as medicines are concerned. Any functional conditions which react on the vascular system must be regulated. The cause of the angina must be discovered and removed if possible. The habits and associations of the individual may be changed with benefit. New associations, travel, change of climate, hydrotherapy, massage, electricity, any or all of these methods as carried out at some good sanitarium, involving a change of scene and associations, may be of immense advantage. Morphine is best to relieve the attacks of angina in the neurasthenic forms, and nitroglycerine is of great use in the vaso-motor forms.

TACHYCARDIA.—Heart hurry is a symptomatic condition of various grades and origin. It is physiological in early life.

a common symptom in the exhaustive period of febrile dis-
eases, in anæmia, and occurs in many diseases of the heart and
blood vessels. The term tachycardia is a relative one, but a
persistent pulse rate of over 120 per minute may be termed
tachycardia. Allbutt criticises the use of the term "tachycar-
dia" to denote various symptomatic states of increased rate of
heart action. He would restrict the term "tachycardia" to the
essential form of the affection, and regards the use of the qual-
ification "paroxysmal" in relation to the latter form as entirely
unnecessary. If, however, it is proper to use the term "spas-
modic" as indicative of a form of asthma essentially different
from other forms of asthmatic manifestations, it is equally
appropriate to make use of the qualifying term "paroxysmal"
as indicative of a form of rapid heart essentially different from
other forms in that, as described by Nothnagel, it comes on
suddenly, lasts a few hours or days, and terminates abruptly.
On the other hand such pedantic use of the term "tachycardia"
as is made by Anders who says that tachycardia may occur
as a physiologic condition in certain people whose pulse may
range from ninety to one hundred beats per minute, seems
somewhat arbitrary. We see at times, however, instances of a
more or less persistent rapid heart in connection with hysteria
and other neurotic states where the pulse-rate may be from
120 to 140 or 160, presenting marked variations in rate but
not the paroxysmal feature peculiar to essential tachycardia
and to which the application of the term "tachycardia" would
seem to be justifiable. In some forms of tachycardia, through
the absence of the usual pause following diastole, there is
equalization of the systole and diastole, the heart sounds are
fœtal-like and tic-tac in character (embryo-cardia). Tachy-
cardia may be of toxic or neurotic origin. Its cause may be a
stimulation of the accelerators or a paralysis of the inhibitory
cardiac-nerve. Balfour thinks that in nearly all cases of tachy-
cardia, myocardial changes are present. Toxic tachycardia
may be caused by tea, coffee or tobacco. (Fræntzel thinks that
tobacco causes changes in the musculo-motor and inhibitory
centers of the heart, which effect is not dependent on the
amount of nicotine.) Belladonna and digitalis cause tachycar-
dia by paralyzing the vagus. Pressure on the vagus by tumors

may cause persistent and fatal tachycardia. Any neurosis, visceral disease or abnormality may cause tachycardia. Persistent tachycardia may be due to toxæmia of gastro-intestinal origin (Thomson).

Recently, I had under treatment a young man with a pulse rate of 160 per minute. This pulse rate had lasted for several months. His heart was healthy and the only thing wrong with him was some gastro-intestinal trouble. He had attacks of dizziness or faintness with pallor of the surface of the body. Regulation of the diet with intestinal disinfection, reduced the pulse rate to 80 per minute within two months.

In a young woman with some nervous trouble of an hysterical type and a continuous high temperature (100 to 102) lasting for months, the pulse rate was from 120 to 140 for over two years, but, with gradual improvement in the general condition, came down to 85 when patient was quiet.

The tachycardia which is one of the cardinal symptoms of Graves' disease, usually partakes of the nature of palpitation, the heart's action being more or less distressing, which is not usually the case in tachycardia, as the patient seldom complains or even notices the heart's action unless suffering from some of the secondary results of continuous rapidity of the organ.

In a young woman of 25 years, with exophthalmic goitre, chronic endocarditis with immense eccentric hypertrophy of the left ventricle, aortic and mitral lesion, the heart rate was from 160 to 200 per minute for over two years that she was under observation, and her history extended over a much longer period. She never complained of her heart and was not disturbed by it unless under great excitement or exertion she should develop palpitation. She married while under observation, had a miscarriage, made a good recovery and did not seem at all worse for her experience in this regard.

Tachycardia is often a feature of mitral stenosis. Balfour says that tachycardia may terminate in mitral stenosis and will disappear with the development of the latter condition.

The treatment of simple or symptomatic tachycardia involves the management of whatever influence of toxic, neurasthenic or reflex nature which may act as a cause. These cases are troublesome and apt to relapse.

Paroxysmal tachycardia (Tachycardia paroxysmalis, synchopexia, essential or recurrent tachycardia, heart-hurry) is a form of rapid heart action (200 or more per minute) coming on suddenly without apparent cause and lasting a few hours or days and terminating spontaneously (Nothnagel). If the attack is short there are no symptoms except the tachycardia. If, however, the attack is prolonged for several days, there may be bloody sputum, the pulmonic and systemic circulation becomes involved. Dyspnœa, rapid breathing, cerebral symptoms, insomnia and delirium appear. The temperature is normal in short attacks but rises 1-3° C. in prolonged attacks (Bouveret). A favorable termination may occur suddenly. The pulse, after a few interuptions of rhythm with energetic contractions, may fall from 200 to 70 beats per minute. The duration of the affection is uncertain. It may extend over years. Each attack is liable to weaken the heart. The longer the duration of the attack the more unfavorable the prognosis. (Bristowe reports a case lasting five weeks, with recovery.) Mental and physical exhaustion seems to be the most prolific cause of the attacks.

Paroxysmal tachycardia was probably first noted by Catton. Watson, Williamson and others soon followed him. The name "tachycardia" was first used by Gerhardt, and the qualification paroxysmal by Bouveret. Among the best descriptions of the condition are those of Martius and of Herringham. Prœbsting gave a detailed description of tachycardia, and Nothnagel described three cases in 1887. Some of the earlier observers appear not to have noted the essential features of the typical form, as, for instance, Fraentzel, who speaks of the "terrifying feeling of palpitation" accompanying the attacks. It will be observed that palpitation is not a feature of the typical attack of essential paroxysmal tachycardia.

Ætiologically the observations of Œttinger appear to establish some hereditary influence. Males appear to be somewhat more frequently affected than females, and the attacks are most likely to appear between the ages of thirty and fifty years, though they may occur at any age. In seven of the fifty-three cases collected by Herringham the affection began in childhood. H. C. Wood, and Watson Williams have each re-

ported instances of tachycardia in patients over eighty years of age. Various diseases have been described in association with tachycardia, though no ætiologic association has been established. Fright, shock, traumatism and various reflex causes have been associated with the onset of the attacks; but the paroxysms generally appear to be, in typical cases, independent of such influences. At the same time, these various influences may unquestionably act as the immediate exciting cause of the paroxysms. Theoretically, the cause has been attributed to either paralysis of the pneumogastric, or to stimulation of the sympathetic nerves. Martius attributed the occurrence of tachycardia to sudden dilatation of the heart. As for disturbance or paralytic inhibition of the vagus, put forth by Tuczek as a cause of tachycardia, none of the results known to follow bulbar disease, pressure on the vagus by tumors or enlarged glands, nor experimental work indicate that such a high rate of action of the heart can be obtained from such sources. Synchronous inhibition of the vagus and stimulation of the accelerators may be admitted as a possible cause but this would postulate some temporary bulbar change, so far impossible of demonstration—possibly a bulbospinal neurosis, as suggested by Debove and Boulay. It may be recalled that Talamon regarded the affection as epileptiform in nature.

Stimulation of the accelerators to the extent of overcoming vagus control, as suggested by Nothnagel, while favored by the character of the onset and termination of the attacks, is opposed by the total inability to produce any like effect by experimental stimulation. Certainly reflex irritations, which in some cases of tachycardia appear to be definite ætiologic factors in the production of individual paroxysms, do not have the inherent power of instituting them. As far as the cardiac ganglia are concerned no definite changes have been demonstrated in them which are associated with tachycardia. Moreover, the researches of Gaskell and others would appear to prove that the ganglia are not sufficiently concerned in the dynamic properties of the heart to produce any such effect as tachycardia. In regard to the effect of changes in blood-pressure from extensive dilatation of the venous or arterial systems, such alterations in the vascular tracts do not produce such a high rate

of heart-beat. In collapse from vaso-motor paresis, from hæmorrhage, or from the sudden removal of large amounts of serous fluid we do not find a pulse rate above 160-180 per minute. In those cases of rapid heart associated with hysteria and kindred states we find the pulse-rate subsiding during sleep or during the administration of an anæsthetic.

Although paroxysmal tachycardia is not associated with any clinically demonstrable disease of the heart, in the six cases which have come to post-mortem there has been shown myocardial disease in three, and if we consider that in those analogous disturbances of cardiac rhythm—tremor cordis and delirium cordis—there is practically always some form of myocardial disease present, we may admit that West's opinion, that the lesion of tachycardia is in the myocardium, is not without some force. Still, the failure to demonstrate some fault in the nerve-supply of the heart does not prove that such does not exist. The great frequency of myocardial disease and the great infrequency of tachycardia show the latter not to be dependent on the former alone. Neverthless, myocardial changes may be necessary, if not a principal factor in the production of tachycardia, at least so far as modifying the inherent automatic and co-ordinate action of the myocardium is concerned. In a heart-muscle which has undergone changes ordinarily not productive of clinical, or possibly even of pathological evidences of disease, a sudden variation of the usual conditions of circulation (ischæmia) or of innervation (toxic causes) or both might be productive of such manifestations as tachycardia.

A typical case of paroxysinal tachycardia is presented in the following history:

Mrs. M., aged 60 years, came to my office in December, 1898, saying she wished to consult me about her heart. She was a well-preserved woman of medium height, and rather stout, with a general appearance of good health. She stated that she had never been ill, with the exception of the ordinary sicknesses of childhood; that she had always been active and energetic in both domestic and social life, but had been obliged within the last two years to be careful in regard to physical

exertion and diet, because of the "spells" with her heart which were becoming more frequent and persistent. She stated that she had had these spells at intervals for several years. At first they were infrequent and of short duration—an hour or two. During the last two years the attacks had become so frequent and persistent as to be present nearly one-third of the time. While she was able to go about and attend to her ordinary duties even during an attack, she realized that she was becoming steadily more and more incapacitated, and this had alarmed her, particularly as she had taken various forms of treatment without any particular benefit.

She stated that she realized when an attack began and when it ended. With the beginning of an attack she felt conscious of a slight fluttering of the heart; during the attack she was restless and nervous, and while conscious that the heart was beating rapidly there was no special feeling of discomfort, while at times she would forget all about it. Usually she slept well, though of late she had difficulty in getting to sleep when the attacks were on. When the attacks ceased she was conscious of a sudden check in the action of the heart and immediately felt relieved as of a nervous strain. She felt exhausted after an attack, in proportion to its length. She stated that she was then suffering from an attack which began on the previous day; that she had been down-town shopping for several hours before coming to see me and was somewhat fatigued. I felt her pulse. The artery was not well filled and the pulse was very compressible. The rate was 210 per minute. The heart-beat coincided with the radial pulse. The machine-like regularity of the heart's action was strikingly characteristic. There was absolutely no variation in the rhythm. The heart sounds were weak and tic-tac in character, the second sound being, perhaps, a trifle higher in pitch than the first sound, though the two were remarkably alike in duration and pitch. There was no subjective or objective evidence of palpitation, tremor cordis, or delirium cordis. There were no cardiac murmurs, and the patient was not dyspnœic. The visible and palpable area of cardiac impulse, was somewhat increased in extent and displaced slightly to the left, but was weak and ill-defined. The left border of dullness was slightly

displaced to the left. The apex impulse remained in the fifth interspace. The second pulmonic and aortic sounds were not intensified.

The patient was seen at her residence the following morning. She stated that the attack had ceased about an hour before my arrival, and that except for her weakness she felt quite well. Her pulse was 70, perfectly regular, and the heart's action apparently normal.

The patient was under observation for three months during which period the attacks became of longer duration and left her weaker and with increasing dilatation of the heart. Upon one occasion I happened to have my fingers on her radial pulse when one of the attacks terminated. There was a sudden cessation of the rapidly-running pulse, a very short pause, a strong, well-defined pulsation, followed immediately by a weaker one, and by the time I could apply my ear to the chest wall the heart was beating steadily at the rate of 70 per minute.

No substantial improvement was obtained by any method of treatment. The attacks increased in frequency and duration until they were on fully half of the time. In the latter part of March, 1899, she had an attack lasting over four days. On the morning of the fifth day I found her much exhausted, dyspnœa, and slightly cyanotic. There were œdematous rales in the base of both lungs; the apex of the heart was outside of the mammillary line; the pulse-rate was 210 per minute but very feeble. It rapidly became imperceptible; the dyspnœa and cyanosis increased and she died quietly with no evidence of suffering. A post-mortem examination was not obtained.

In contrast with the above case I may cite a case of paroxysmal tachycardia probably from reflex causes. It occurred in a man, the subject of relapsing typhoid. His pulse had been very steady and not at any time above 90 per minute. During the sixth week of the fever his pulse suddenly jumped to 260 per minute, was perfectly regular, and typical of the pulse of paroxysmal tachycardia. A saline and an enema were followed by a free evacuation of the bowels, after which his pulse dropped abruptly to 70 per minute. The patient made a good recovery.

The essential features of paroxysmal tachycardia are: the sudden onset, the great rapidity of the heart's action, its machine-like regularity, the absence of palpitation and dyspnœa, and the abrupt ending of the paroxysm. Such subjective sensations as pain in the chest, uneasiness, numbness, tingling, or consciousness of motor disturbance of the heart may be present, but usually are not. Indeed their absence is rather one of the differential features between essential and symptomatic tachycardia. Rarely there may be carotid or jugular pulsation, but as a rule these, like dyspnœa and cyanosis, are associated only with the cardiac dilatation which betokens the imminence of dissolution.

The prognosis is unfavorable in recurrent cases, and in all cases is uncertain. Recurrence usually becomes more frequent and the attack prolonged until fatal dilatation of the heart occurs. Herringham's statement that the prognosis depends on the frequency and duration of the attacks, is true enough, but his remark that few patients pass the age of fifty years, is not substantiated by the records. In some instances the attacks have grown more infrequent as the ages of the patients advanced.

The treatment of paroxysmal tachycardia is unsatisfactory. There being no specific indications, naturally the therapeutic management of these cases must be empirical, aside from the usual means applicable to any abnormal condition of the heart muscle, or to any eccentric factors of which observation in the interval of the attacks may indicate some possible casual relationship. All the hygienic and other measures adapted to the management of cardiopathic states in general are applicable in this connection; for even if no abnormal condition of the heart can be clinically made out we can not assume that the heart is healthy. The diet and exercise should be adjusted as in other cardiopathies. Eccentric sources of irritation should be searched for, especially in the intestinal tract. Auto-toxic conditions should be avoided. During the attacks morphine may be used in some cases, though as a rule all vaso-dilators are contraindicated. If dilatation of the heart is increasing, digitalis should be cautiously given. As a rule digitalis does not influence the pulse-rate for the better and

its utility is doubtful. Cupping or leeches may be useful where there is pulmonary stasis. Stimulation of the vagi by compression (Bouveret) or electricity to the cervical portion is recommended, but has utterly failed at my hands. According to Brieger electricity to the præcordium, vagus compression, and stimulants have produced slowing of the heart's action. Rosenfeld recommends compression of the chest. It may be practiced by the patient, who fixes the feet against the bottom of the bed, applies the arms firmly to the side of the chest and the fore arms to the lower anterior portion of the chest wall; inspires deeply, fixes the glottis, and makes strong expiratory efforts while pressing firmly against the chest with his arms.

Massage of the heart through the fourth and fifth interspaces seemed to me to increase the force of the heart's action, but not to cause any change in the rate. Ice-bags to the præcordium are recommended, but I have seen no benefit from their use.

Poulet recommends coronilla scorpioides, particularly in cases associated with valvular disease. Balfour suggests an ether spray to the cervical spine or chloroform poultice over the præcordium.

BRADYCARDIA (*pulsus rarus*).—The term bradycardia is applied to a permanent form of slow pulse. This must be distinguished from the so-called false bradycardia due to a varying number of hemosystolic actions of insufficient force to reach the radial arteries, the pulse assuming the form of the pulsus alternans, bigeminus or trigeminus. In the sphygmogram of false bradycardia there may be, half way down the long descending limb, a slight elevation just where the percussion wave of another pulsation should begin, while in true bradycardia the tracing has an anacrotic appearance. False bradycardia is a frequent form of the arrhythmia which appears in the ataxic period of cardiac lesions. Slow pulse occurs from irritation of the vagus, paralysis of the sympathetic, disturbance of the cardiac centers, in the exhaustion of convalescence, in meningitis from pressure on the brain, in jaundice from the toxic action of the biliary acids, in epilepsy, in colic, in stenosis of the aortic or mitral valves, in various degrees of

coronary sclerosis, from injury of the cervical portion of the cord, and from the action of digitalis. Balfour thinks that slow pulse is not due to any cardiac lesion but to cervical inhibition acting through the accessory nerve, the possibility of an idio-ventricular rhythm from vagus inhibition arguing against the latter condition as a cause for slow pulse. Huchard thinks the majority of cases due to sclerosis of the coronary arteries and to a true bulbar ischæmia. Prentis, after analyzing ninety-six cases, concludes that slow pulse may be produced by disease or injury of the nerve centers; of the pneumogastric or sympathetic nerves; of the cardiac ganglia; of the heart muscle; by the action of poisons or toxines. In view of the probable myogenic nature of the cardiac action the possible role played by the various toxines in causing the rhythmic disturbances of the heart, is an interesting question, especially when we consider that no definite pathological condition of either the heart muscle, arteries or nervous mechanism can be constantly associated with abnormalities of the heart's rhythm.

Bradycardia is a relative term, as a pulse of forty may be compatible with good health. Usually a pulse rate under fifty may be termed a bradycardia. Slow pulse occurs more often in men than in women, and, while found in early life, it is more frequently an accompaniment to senility. The pulse rate may get as low as eight or ten per minute, though from twenty to thirty is a much more common rate for a permanently slow pulse. The patient does not appear to suffer in any way from the slow pulse unless it is accompanied by some irregularity, as delirium cordis. Permanent slow pulse may last for a lifetime, or the pulse may become normal after months or years. Usually after a few years, epileptiform attacks occur and in one of these attacks the patient succumbs.

The treatment of bradycardia is purely symptomatic. In cases associated with vascular sclerosis one may follow Huchard's advice and give nitroglycerine, or the iodides of potash or sodium. In false bradycardia, digitalis and other heart stimulants are indicated. In true bradycardia, digitalis is permissible when the vascular tension is low and the pulse flat and ribbon-like when it may be very useful, or, with muscular weakness and high tension, digitalis may be carefully used in

connection with vaso-dilators. Belladonna will increase the pulse rate, but should be used cautiously. Douglas Powell advises caffein in bradycardia with scanty urine. In those cases of bradycardia which are associated with myxœdema or which follow Graves' disease, influenza or diphtheria, the treatment is modified with reference to the previous or associated conditions.

CARDIAC ARRHYTHMIA.—There are many varieties of irregularity of the heart's action. In the majority of instances the irregularity is not of any special type and is an evidence of temporary disturbance of innervation or of muscular inability, actual or relative. In some instances the irregularity will assume a definite form—a regular arrhythmia (allorrhythmia). In this way we have recognized types of pulse rhythm such as the *pulsus alternans* (two heart beats to one pulse, or second pulse is feeble); *p. bigeminus* (every third beat omitted); *p. trigeminus* (every fourth pulsation omitted), etc. The cause of arrhythmia may rest in myocardial disease, or in nonsynchronous contraction of the ventricles and auricles through vagus influences. Heitler has demonstrated that in the dog mechanical or electrical stimulation of the visceral pericardium disturbs the cardiac rhythm even after section of all the cardiac nerves, except when the heart is painted with cocaine. The excitable regions are: The upper third of the longitudinal sulcus, the left ventricle over the upper third, and the right ventricle close to the sulcus. The excitability of the apex varies. Huchard recognizes three kinds of arrhythmia: myocardial, nervous, and mechanical. Arrhythmia may be of psychical, cerebral, nervous, toxic, reflex, or cardiopathic origin. The specific relations of arrhythmia are too numerous to be individually considered. There is an irregularity of senile life, coming on very gradually and without any disturbance, it is probably due to myocardial degeneration. Arrhythmia in infants is considered as indicative of serious nervous troubles by Richardson, and of myocardial disease by Balfour.

There are special forms of arrhythmic heart action which are worth a moment's separate consideration.

Palpitation. By palpitation we mean a sufficient increase in the force and frequency of the heart to cause distress to the

patient. The pulsation is regular, rapid and violent, and is accompanied by a throbbing of the larger arteries which does not extend to the smaller vessels. Palpitation is more apt to occur in young than in old people, and is supposed to be due to reflex inhibition of the vagus. Palpitation occurs most often in anæmic individuals, from reflex gastro-intestinal causes, emotional disturbances especially during the climacteric, and from many of the causes which also produce irregularity. [The rapid, forcible heart-action produced by exercise, particularly in anæmic persons, is very similar to palpitation but is produced by increased action of the augmentor nerve, the radial pulse being increased in force and volume. Rest immediately relieves this condition.] In some cases there seems to be an hereditary neurotic element in the causation.

There is a recurrent form of palpitation directly or indirectly associated with acute infectious diseases exhibiting a variety of symptoms in connection with the general nervous or vaso-motor systems which Sansom calls "vagus storms." He thinks these symptoms due to "morbid change or recurrent irritation of the nerve elements in the medulla oblongata and the spinal cord," and recommends Fowler's solution for their control.

The treatment of palpitation is entirely symptomatic. In many cases sedation with sodium bromide and aconite is advisable.

Tremor Cordis. There is a very interesting form of irregularity of the heart in which the heart, while beating in the usual rhythm, suddenly and without apparent cause, takes on a very rapid, tremulous, fluttering action which varies from a few rapid, incomplete systoles to a continuous tremor lasting ten or fifteen seconds and ending suddenly like an intermission of the pulse, with a forcible beat, the heart then resuming the usual rhythm. This form of arrhythmia may occur with an otherwise regular pulse or it may be associated with other forms of irregularity of the heart.

Tremor cordis occurs at any time of life but is most common after middle age in persons with weak, dilated hearts, particularly in association with gastric disturbances or flatulence. Emotional disturbances do not seem to produce tremor

cordis. It occurs with apparently healthy hearts or in connection with any form of cardiac lesion. During the tremor the pulse at the wrist may be imperceptible, or a faint tremulousness of the artery may be felt. Palpation of the præcordium shows an indistinct trembling action of the heart. Faintness or syncope do not seem to accompany the change in the pulse rate. When tremor occurs in a heart which has been beating regularly, the patient will be made aware of it by an uneasy sensation in the chest which may frighten him but which passes away with the cessation of the tremor. When tremor is incident to some more constant form of cardiac arrhythmia, the patient may be entirely unconscious of its occurrence. Balfour thinks tremor cordis is due to vagus inhibition with gradual overfilling of the ventricles. Sudden exercise of augmentor power empties the ventricles with forcible action and allows the heart to resume its usual rhythm.

Delirium Cordis. Delirium cordis is an irregularity of the heart in which there is a number of rapid beats huddled together in inextricable confusion. This jumble of beats will end abruptly and be followed by a short silence, then one or two ordinary but forcible contractions, and then another series of delirious contractions in which first and second sounds are altogether confused and the pulse wave is a series of rapid percussion strokes of very great variation in force. The sphygmogram shows the remarkable variation in force of the radial pulse. This peculiar rhythm is supposed to be due to independent ventricular and auricular action. At times when the two coincide, the slow beats occur.

Delirium cordis occurs frequently in mitral stenosis and in dilated hearts of gouty origin, also in myocardial degenerations associated with chronic rheumatic arthritis. It may occur merely as a temporary phenomenon or it may be a permanent affair. It is startling at times, to feel the pulse of a person who is well to all appearances and find it tumbling around with such remarkable irregularity, yet the subjective symptoms are unimportant though the subject is usually aware of the irregularity in the pulse. Delirium cordis sometimes appears during the paroxysm of bronchial asthma. It may occur when digitalis acts unfavorably or is not tolerated, and

it is sometimes the herald of the approach of asystolism. The treatment of delirium cordis includes careful diet, antarthritics, general tonics, antacids and in some cases cardiac stimulants.

The following case presented an interesting combination of tremor cordis with delirium cordis.

Woman aged 50. Had always been in good health and of remarkable powers of endurance. Had always been much in public life and for the last three or four years her public duties had been continuous and exacting. Had not been well for a month and made considerable effort to keep up with her work. For a week has had some irregularity of the heart which was not particularly troublesome. Last night had a sinking spell accompanied by dyspnœa and considerable distress in the region of the heart. I saw her towards morning about five hours after the attack began. She was flushed though the surface of the body was cool and the peripheral circulation was poor. There was moderate dyspnœa which was increased by movement. Considerable pain about præcordium with a painful consciousness of the heart's action. There had been no loss of consciousness.

The heart was beating over 200 per minute and was so irregular that it could not be counted with the aid of a stethoscope. The pulse was so weak at times that is could hardly be felt. There was gastric distress and flatulent distention of the abdomen.

Under treatment with digitalin and strychnia the pulse improved and the rate came down to 160 per minute and was not so irregular. About once in every 45 to 60 seconds the irregularity would assume the form of a tremor cordis lasting about eight or ten seconds when the usual pulse would reappear for half a minute or more to be again interrupted by tremor of the heart. The patient was not conscious of any difference in the heart's action during the period of tremor though the pulse at the wrist was merely a tremulousness of the artery, if it could be felt at all.

There was increased force and area of cardiac pulsation except during the tremor when little or no motion could be

felt. The heart was slightly dilated. There was no valvular lesion.

The patient gradually recovered so as to be about with a fairly regular heart, and was lost sight of.

Gallop Rhythm. This is a peculiar rhythm of the heart sounds which resembles the sounds produced by the hoofs of a galloping horse. There are three sounds to each heart action. In true gallop rhythm two of these sounds are diastolic and the first diastolic sound is accentuated (Fraentzel). Various descriptions have been given of the gallop rhythm. Traube described a "sort of gallop rhythm," and Potain, Johnston and Barie described it as a reduplication of the first sound. Fraentzel's distinction as to the character of the rhythm is more in conformity with its clinical manifestations. He admits the doubtful nature of its development, but assumes that it is due to weak heart action, reflux of blood in the two arterial tracts at different times with a corresponding difference in the time of production of the second sound in the two arterial tracts. There must be some other causative element than weak heart, for gallop rhythm is not common in hearts that are merely weak.

Gallop rhythm occurs in chronic nephritis (interstitial), in some of the so-called idiopathic cardiopathies, in acute and chronic anæmia, Addison's disease (Lagus, Fraentzel), typhoid fever (second or third week, indicative of heart failure), pneumonia (at or about the crisis, a sign of impending collapse), typhus fever, diphtheria, acute rheumatism, acute milliary tuberculosis, etc. Though gallop rhythm appears to be entirely independent of any demonstrable heart lesion, it is nevertheless an evidence of weakness of the heart, and in acute diseases, at least, is an indication for the exhibition of heart stimulants.

In the following case gallop rhythm developed in a cardiopathic patient suffering from influenza infection.

Woman aged 42. Chronic valvulitis (aortic regurgitation and mitral stenosis) with good compensation. Influenza with gastro-intestinal manifestations eventuating in a typhoid state. (Widal's test, etc., excluded typhoid fever.) The urine

contained some granular casts. The total solids about nor-
mal. Temperature from 97°F to 101.5°F. Pulse irregular,
very weak at times. First two days pulse 160-180 per min-
ute; fourth day fell to 38 per minute; varied from 40 to 58
for three days, then rose to 70-78, always irregular. There
were spells of restlessness with dilatation of peripheral vessels
followed by attacks of unconsciousness lasting from 2 to 10-12
hours. Gastric irritability and tympanitis were troublesome.
On tenth day a well marked gallop rhythm developed. The
accentuation of the first diastolic sound was well defined. The
pulse at the time was 73 per minute. An attack of uncon-
sciousness on the twelfth day was followed by death in six
hours.

CHAPTER VIII.

BRONCHITIS.

Inflammations of the bronchial tract have been classified from ætiological, pathological and topographical standpoints by various observers. These distinctions, while serving to individualize special types of the disease, are more or less confusing. The simplest way is to restrict the classification to those well marked clinical types which, though they may at times exhibit unusual features, are still sufficiently constant in their course to constitute definite forms of bronchial inflammation.

Thus we find that bronchitis may be acute or chronic.

Acute bronchitis presents three distinct forms: *acute catarrhal* (involving the larger tubes); *bronchiolitis* (capillary bronchitis, suffocative catarrh), and *fibrinous bronchitis* (croupous bronchitis, plastic bronchitis).

The unqualified term, acute bronchitis, applies to catarrhal inflammation involving only the larger and medium sized tubes. [The term bronchiolitis is used as entirely distinct from broncho-pneumonia. The attempt to unify these conditions clinically (Legendre, Rilliet, Roger and others) is a mistake, although it is admitted that the morbid anatomy of the two conditions is intimately associated. Nearly all fatal cases of bronchiolitis have resulted in atelectasis or in lobular consolidation. Autopsies, therefore, show the presence of these conditions, nevertheless, bronchiolitis is a distinct affection, and while the clinical divisions of Germain Sée, of diffuse bronchitis of the finer tubes; bronchiolitis of the intralobular branches; and broncho-alveolitis of the alveolar bronchi, appear somewhat more subtle than the possibilities of clinical diagnosis would warrant, we still believe in adhering to the principles of Laennec in recognizing clinically a distinct affection of the bronchioles.]

Bronchitis is most common in infancy and in senile life, because of minor degrees of individual resistance. Dentition and abnormalities of the nasal and pharyngeal passages are frequent factors in producing bronchial inflammation. Age, sex, occupation, physical condition and habits influence the occurrence of bronchitis so far as they induce added exposure and lessened resistance to the influences which determine bronchial inflammation. There is a marked feature of heredity in some forms of recurrent asthmatic bronchitis and in some cases of winter cough. Cachexia of gouty, syphilitic, tubercular, alcoholic or nephritic origin predispose to bronchitis. Spinal curvature and other deformities of the chest predispose to bronchial inflammation.

ÆTIOLOGY.—*Acute bronchitis* may result from the topical effect of cold air on the skin or mucous membranes of the air passages. This is difficult to reconcile with the clinical fact that bzronchial catarrh is more apt to result from a partial or limited, than a general, exposure to cold. According to Thomson, bronchitis seldom results from some irritant property or ingredient of the inspired air acting on the bronchial mucous membrane. Primary bronchitis occurs with such contagions and infections as influenza, measles and whooping-cough. Acute bronchitis may be secondary to small-pox; also from general or cardiac weakness incident to typhoid fever. Cachectic and septic conditions may give rise to acute secondary bronchitis. Local irritations from dust, gases or chemicals (iodine, bromine) may cause acute bronchial catarrh.

Bronchiolitis is especially an affection of infancy and senile life. It may be primary (one-third of the cases—Roger) or secondary, general or localized. All the causes of acute bronchitis of the larger tubes may likewise cause bronchiolitis. In a large proportion of cases it results in extension of the former condition to the smaller tubes, especially in the infectious diseases of childhood, although in many of these cases it is primary. The influenza infections are especially prone to induce bronchitis, which is frequently localized particularly in the lower portions of the lungs. Measles and whooping-cough are the most frequent causes of bronchiolitis

before the age of five years. Emphysematous conditions, chest deformities, rachitis, spinal curvature and all conditions which modify respiratory power, especially in young and weak children, are contributory toward the occurrence of bronchiolitis.

Fibrinous bronchitis is a rare affection. It occurs most often in males (two to one—Fowler), and between the ages of fifteen and forty-five, although it may occur at any age. Occupations entailing exposure to cold and wet predispose to its occurrence, and the associated conditions are pulmonary tuberculosis, valvular disease, pneumonia, typhoid, cutaneous diseases, pregnancy and the catamenia. These conditions are, however, accidental, and the ætiology of fibrinous bronchitis is not known.

Chronic bronchitis occurs most frequently in old age or in early life. It may be primary from exposure or from the inhalation of irritating or poisonous air, especially when there is some constitutional vice. Secondary chronic bronchitis occurs most frequently as a result of acute attacks of bronchitis. Climatic conditions, senility, emphysema, alcoholism, Bright's disease, gout, psoriasis and eczematoses. Mitral lesions, pulmonary tuberculosis, the pressure from aneurisms or mediastinal growths, bronchial stenosis or dilatation, are some of the conditions which induce chronic bronchial inflammations.

MORBID ANATOMY.—In *acute bronchitis* there is hyperæmia of the inner fibrous coat which becomes swollen and infiltrated with lymph cells. The basement membrane becomes œdematous and irregular in appearance. The ciliated columnar epithelium becomes detached in patches, and from the deeper layer of flat cells various transitional forms of cells are produced and thrown off with quantities of leucocytes. All the structures of the bronchial wall become involved and leucocytes infiltrate the muscular and fibrous coats. The secretion of the mucous glands of the bronchi is greatly increased, the epithelial and secreting elements of the glands becoming desquamated. On post-mortem examination the bronchial lining may not appear much altered owing to the action of the elastic fibres on the blood vessels. In some cases it may be reddened in patches. The bronchi may contain clear, yellowish or greenish mucus. Obstruction of the tubes may pro-

duce distention of the alveoli. Pulmonary congestion, œdema, or atelectasis may occur. Emphysema of the surface or borders of the lungs develops. The right heart is overtaxed and ultimately becomes dilated.

Bronchiolitis produces changes similar to acute bronchitis. Its greater gravity depends on the interference with respiration from obstruction of a large number of the smaller tubes, and from the weakened condition of the bronchial muscles, especially in children.

The lining of the tubes is markedly injected. The tubes may be blocked with secretion and small beads of pus can be expressed from the smaller tubes. Pulmonary emphysema, atelectasis, œdema, and small, yellow areas of bronchopneumonia may be present. The bronchi may be more or less irregularly dilated. The right heart may be dilated and venous obstruction is greater than in acute bronchitis. Bronchiolitis is more often a general than a local affection.

Fibrinous bronchitis is entirely distinct from diphtheria of the bronchi. Its exact pathological nature is not known. Its dependence on any specific microbe has not been demonstrated. The exudation may be limited to a few tubes or may be scattered through numerous areas in both lungs. The exudation forms a complete cast of the tubes affected, but does not block the larger tubes, though the smaller bronchi may be occluded with cylindrical, solid casts. The expectorated casts vary in size from small pieces, or branches, to complete casts of a bronchial tree from two to six or seven inches long. Their minute subdivisions may end in a bulbous extremity which may correspond to an infundibulum (Biermer). The casts are white or gray in color, of firm consistence, and may be flecked or stained with blood. The larger branches have nodular prominences. A cross-section of the larger casts shows a central lumen of more or less size and a lamellar structure of the walls, which consist of concentric laminæ of fibrin enclosing in its meshes leucocytes, mucous and epithelial cells. Charcot's crystals, pigment cells, fat globules and blood corpuscles may also be present. [Recently it has been claimed by Beschorner, Neelson and Grandy that the exudation in fibrinous bronchitis consists of mucin and not of fibrin. Klebs

and Eppinger maintain the fibrinous nature of the casts. In two specimens recently obtained by me, a careful series of tests made by Prof. Herzog, determined the casts to be of a fibrinous nature.]

The trachea is seldom or never affected in this form of bronchitis. After death the bronchial lining may be intact and pale, or may be congested, or may be absent. The bronchial wall may be inflamed. The lung may be emphysematous or atelectatic.

Chronic bronchitis affects any portion of the bronchial tract. It is usually limited to the larger and medium-sized tubes. The bronchial membrane may be deeply injected and of a purple or violet color. In the larger tubes longitudinal layers may be formed by the elastic fibres of the inner fibrous coat. The tubes are narrowed or dilated (bronchiectasis). The bronchial glands are enlarged and black in color. Emphysema of the peripheral portions of the lungs is usually present. Congestion and œdema of the base of the lungs may occur and the heart may be hypertrophied or dilated.

The ciliated epithelium disappears in places and is replaced by transitional cells. Dilatation of the blood vessels and cellular infiltration cause great thickening of the inner fibrous coat. Various stages of hypertrophy and atrophy of the muscular coat will occur from the infiltration of the bronchial wall or from dilatation of the tube. Peribronchitis is present to a greater or less extent, especially at the periphery of the lung. The cartilages may be normal, softened or calcified. The mucous glands are distended or destroyed. The muscular coat of the small arteries is hypertrophied and the capillary vessels dilated. Diverticula of the mucous membrane may form through relaxation and fissures in the middle coat. Induration of surrounding lung tissue occurs. Ulceration of the bronchial lining is rare, but may occur in tuberculosis and in very old people. Fistulous communications may form. In tertiary syphilis, gummy tumors and fibrous stenosis of the bronchi may occur. Fœtid, putrid expectoration is generally associated with bronchiectasis, though it may occur without it. The decomposition of the secretion may cause no trouble or it may cause gangrene of the lungs.

CLINICAL HISTORY.—The clinical history of *acute bronchitis* extends over a variable period lasting from a few days to three or four weeks. Its duration depends largely on the general resistance of the subject and on the condition of the respiratory apparatus; pulmonary emphysema, induration, pleuritic adhesions, etc., prolonging its cause considerably. There may be a history of a common coryza, pharyngitis and tracheitis preceding the bronchial symptoms, or the latter may come on suddenly without these premonitory conditions, especially in children and old people, or where there has been previously successive attacks. A history embracing any of the ætiological factors of the disease may be obtained. An initial chilliness may occur, but rigors are rare. The temperature is seldom above 101° F., and the pulse is only slightly increased in rate. General malaise and aching may be present. The special features are pain, cough, expectoration and dyspnœa. The pain is an early symptom and is usually a substernal soreness, or it may extend across the front of the chest, or, in localized bronchitis, be located in any portion of the chest. The patient indicates the seat of the pain with the flat of the hand and not with the finger-tips, thus distinguishing it from the lancinating pain of pleurisy.

The cough begins with the onset of the disease. It is first dry and hacking and may be paroxysmal. It is painful at first, and there is subsequent soreness about the sides of the chest along the attachment of the diaphragm. The most sensitive portion of the bronchial tract is at the bifurcation of the bronchi (Nothnagel), though all portions are sufficiently sensitive to cause cough except the smaller branches where accumulation of mucus may result only in dyspnœa. With the appearance of secretion the cough is not so painful, is more connected and is performed with the deliberate intent of promoting expectoration. The expectoration is at first viscid and tenacious, but as secretion increases it becomes frothy and may be streaked with blood. Later the sputum becomes more watery or else more turbid and yellowish, which indicates recovery. In the early stage the sputum consists of clear fluid, ciliated and cylindrical epithelium and mucous corpuscles; later of large numbers of pus cells, epithelial cells, hyaline

cells, granular matter, oil globules, pigment granules and blood corpuscles.

Dyspnœa is usually not marked in a primary attack in an adult. The breathing may be difficult, *labored*, but is not much accelerated. In children, however, the respiratory rate may be much increased. Moderate obstruction of the tubes may produce much dyspnœa if previous attacks have modified the functional ability of the lungs.

Bronchiolitis is a serious malady. Its mortality is variously given as from thirty to fifty per cent. In the majority of instances it is secondary to acute bronchitis of the larger tubes or to the infectious diseases. It is peculiarly an affection of infancy or old age. Whether it be primary or secondary, its advent is announced by a rise in temperature of from two to three degrees. There may be a chill, and in children convulsions may occur. There is frequent paroxysmal cough and pain. In children there is drowsiness, great dyspnœa, the nostrils are dilated, the respiration is hurried, the face hot and flushed. The cough is whistling or wheezing in character, the pulse is rapid and the temperature 102-103° F. The pulse gradually rises to 150-180 per minute, and the temperature to 104-105° F. in the evening, although, as a rule, it is not as high as 104° unless pneumonia be present. The respiration may be from sixty to ninety per minute. There is great restlessness and signs of exhaustion. Delirium and coma may appear, followed by death from asphyxia, preceded by blue lips, pale or livid and cold face, thready pulse and cessation of cough.

The duration of bronchiolitis varies from one to three weeks. Recovery occupies a longer time. Fibroid induration of the lungs extending to the pleura may result. Bronchial dilatation may occur in these indurated areas with surrounding emphysema. There may be continued muco-purulent expectoration. These cases may be mistaken for tubercular disease.

The following history is from a case of bronchiolitis complicating chronic bronchitis in an old person:

Mrs. S., aged 71. Has had chronic bronchitis for forty

years. Weight about 90 pounds. Bronchial tubes in right lung dilated. Expectorates about a pint of mucus in 24 hours and has done so for years. Feb. 12th, '98, had some pain in right chest, considerably more dyspnœa than usual. Temperature rose to 103° F. Pulse 130. Expansion diminished on right side. Respiratory sounds very feeble, percussion pitch slightly raised, no consolidation, rales fine and crepitating on same side. Very little air getting into lower part of right lung. Skin cold and surface somewhat cyanosed, expectoration diminished. Comatose part of the time. Treatment: Five grains of ammon. muriate and half teaspoonful of liq. am. actetat. every two hours; 1/20 grain of strychnia every two hours; small doses of whisky occasionally. Mouth wash of peroxide of hydrogen to cut viscid sputa from throat. Recovery in three weeks.

Fibrinous Bronchitis presents a class of cases exhibiting but one or two attacks (acute), and these may be separated by years, but having had one attack there is a liability to recurrence. In another class (chronic) there is a succession of attacks, usually of a lesser grade of severity, the intervals presenting an indifferent chain of bronchial symptoms.

The clinical picture of the so-called acute form is represented in the following histories:

Male, aged 40, married, beer-bottler by occupation, was perfectly well until two years ago, when he caught cold while working in a wet place. During last two years has had repeated attacks of bronchitis. These attacks would last a week or ten days and were characterized by a severe, rasping cough. Expectoration was comparatively free and there was no particular pain in the chest. About ten weeks before his recent attack he began to cough and expectorate large masses of tough, tenacious sputum, which was accompanied at times by some bloody expectoration. About four weeks ago had high fever, great dyspnœa and distressing cough. There was diminished expansion and dullness over the lower two-thirds of the left lung as compared with the right lung. This condition lasted three or four weeks during which time he was confined to bed

suffering from dyspnœa, cough, and a temperature of above 102° F. Near the end of the third week he expectorated considerable blood. The temperature remained high and the dyspnœa and cough distressing. Three days afterward he had a severe attack of dyspnœa and cough. The temperature rose to 104° F. After considerable effort he expectorated a large mass of material consisting of a cast of the bronchi mixed with mucus and some blood. The temperature became

immediately reduced and the dyspnœa relieved. Examination, a short time after the cast was expelled, showed the air to be entering all parts of the left lung with comparative freedom. The percussion note was almost normal and mucous rales were to be heard all over both lungs. Improvement was rapid.

The cast was gray with a tinge of brownish red. Total length 10 cm. Two stems: the shortest about 1 cm. long and about as thick as a lead-pencil. The branches run out to fine filaments with terminal dilatations. The larger tubes have nodular prominences.

The following history with a specimen of the cast was furnished me by Dr. R. Broughton, of Dwight, Ill.

Woman, aged 34, weight 120 pounds. Mother of two children. Anæmic. Has had several attacks of what was supposed to be *la grippe*. On Nov. 21st, '97, complained of headache, followed by a chill, then fever from 99 to 104° F. for about a week. Very little expectoration, though she had severe coughing spells. On Nov. 29th, after a severe and exhausting coughing spell, she expectorated a well-marked tube cast. Three other casts were expelled at intervals of from three to four days. Following expulsion of the cast the ex-

pectoration was frequent and bloody, though not great in quantity. After the last cast was expelled the blood ceased. Death Dec. 9th. No autopsy.

In some of the "chronic" cases, small portions of casts may be expectorated for days or weeks at a time. Pneumonia is not an infrequent complication of the more severe cases, and may be the cause of death. Fagge reports a death by asphyxiation from lodgement of a portion of a cast at the tracheal bifurcation.

Chronic bronchitis is met with in its mildest form in the winter cough of middle-aged or elderly people, which gradually becomes a constant affair. It may occur as a result of

a succession of acute attacks and is marked by its proneness to relapse.

The cough gradually becomes continuous until the disease is marked by remissions rather than by intermissions during its most favorable periods. The general bodily strength and weight may be maintained for years against this disease, but eventually becomes compromised, and dyspnœa upon exertion becomes associated with asthmatic breathing. Finally dyspnœa becomes permanent as a result of weakness of the right ventricle whose hypertrophy has hitherto offset the resistance to the pulmonary circulation entailed by the changes in the pulmonary parenchyma.

The cough of chronic bronchitis exhibits great variety in character, and in the quantity and quality of the expectoration. One variety is characterized by very viscid, pearly, scanty expectoration (dry catarrh), with violent and rapid paroxysms of coughing which may induce vomiting. The mucus is dislodged and raised with difficulty and emphysema is likely to develop in this form of bronchitis. Gouty arthritis is likely to be associated with this form of dry catarrh. Again, in gouty subjects with weak hearts, we may have more sudden attacks resembling pulmonary congestion and cardiac dyspnœa, with free expectoration.

In most cases of chronic bronchitis, two or three spells of coughing during the day will be attended with quiet intervals. In others, there is constant expectoration often depending largely on cardiac weakness. In some cases, particularly in old people, the amount of expectoration may be one or two pints in twenty-four hours (bronchorrhœa). It may be liquid and frothy (bronchorrhœa serosa—Biermer) or may be yellowish, or like the white of egg. Laennec described a form of bronchorrhœa, under the title "Chronic Idiopathic Pituitous Catarrh."

In the chronic bronchitis accompanying syphilitic lesions of the bronchi, the catarrh may be general when associated with secondary lesions, or localized when in connection with the tertiary forms. Bronchial stenosis or dilatation are usually responsible for the profuse fœtid expectoration. Pain, emaciation and night sweats may be present in these cases.

Fœtid or putrid expectoration is usually associated with bronchiectasis, stenosis, or with pulmonary gangrene, but may be present without either of these conditions. The breath may remain offensive after the expectoration has lost its odor. Again, the fetor may come and go. Some of the worst cases do not appear to be connected with any cause in the tubes or lung tissue. Attacks of lobular pneumonia are frequently a part of the clinical history of chronic bronchitis in both children and old people, and in the latter are particularly to be feared.

SYMPTOMS AND DIAGNOSIS.—In *acute bronchitis* lateral expansion is often deficient, and in children there may be inspiratory recession of the interspaces and ribs at the sides of the chest. The patient, during paroxysms of painful cough, assumes an upright position, leaning straight forward, which is indicative of a bilateral source of the trouble and distinguishes it from pleurisy. Palpation is negative unless mucus in the larger tubes should produce a rhonchal fremitus. The percussion note is unchanged or is hyperresonant from emphysematous conditions. Dullness at the base suggests pulmonary congestion, œdema, or collapse. Auscultation shows harsh, rough breathing, with some prolongation of the expiratory sound. Sibilant, wheezy respiration is present during the dry stage. With the stage of secretion there appears sonorous rhonchi in the larger tubes, large and small mucous rales in the medium sized tubes, and sub-crepitant rales in the smaller tubes. These rales are heard best during inspiration, but are also heard with expiration. They may be changed by coughing. The sibilant rales are not apt to change on coughing, as they are due to narrowing of the smaller tubes. The fine, sub-crepitant rales are heard just *before* the immediate end of the inspiratory act and differ from the crepitant rale of pneumonia by being altered by coughing and are heard at times during expiration. The pneumonia rale is heard *at* the end of inspiration, is not heard in expiration, is followed by a clear, high pitched, expiratory sound and is not modified by coughing or respiration. Pleuritic friction may resemble the rales of bronchitis but occurs earlier in the inspiratory act than a rale of the same size and character, and is not changed by

cough or respiration. Cardiac failure from mitral stenosis, may give rise to a congestion of the lungs easily mistaken for bronchitis, as the mitral murmur may disappear with the advent of cardiac failure (Broadbent). The effect of digitalis or perhaps of venesection, with the reappearance of the murmur, will establish the nature of the trouble.

Miliary tuberculosis has a higher temperature range than acute bronchitis, and more prostration. Apical bronchitis must always be regarded as of probable tubercular origin. Localized bronchitis in the lower part of the lungs is a frequent result of influenza infections, the usual auscultatory signs being confined to a small area, the upper portions being clear.

Bronchiolitis changes the type of breathing, especially in children. The upper portion of the chest scarcely moves and is distended with residual air, while the lower ribs, intercostal spaces and epigastrium show marked inspiratory recession. The percussion note is unchanged in the lower portions, or may be dull from collapse or œdema. In the apices there is hyper-resonance.

Auscultation, early in the disease, shows good respiratory sounds in the upper chest, but feeble sounds below. Later, when distention of the upper chest occurs and in old people with emphysema, the breath sounds will be generally feeble. Any variety of rales may be present, but in the lower portions of the lungs, especially posteriorly, we hear the very fine, crackling rales characteristic of bronchiolitis. They are not so finely crepitant as the pneumonic rale, nor as bubbling as the œdematous rale. The distribution of the rales determines the extent, generalization or localization of the affection.

Bronchiolitis is distinguished from acute bronchitis by higher temperature, greater difficulty in breathing, more dyspnœa, poor circulation and the character of the rales; from pneumonia, by absence of initial chill, of pain in the side, of panting character of respiration, of pneumonic sputum, of signs of consolidation. The temperature in pneumonia is continuously higher than in bronchiolitis. Pulmonary œdema is distinguished from bronchiolitis by its associations, and location in the lowest part of the lungs resulting from blood stasis.

Miliary tuberculosis in children, and acute pneumonic tuberculosis may resemble bronchiolitis. The early apical signs in the former, and the higher and more continued fever in the latter, as well as its location in the upper lobes, will differentiate these conditions, at least with the aid of the history, associations and sputum examination.

Fibrinous bronchitis is usually not recognized prior to the expectoration of an exudate. After this has occurred signs of bronchial catarrh in more or less limited areas may lead to a provisional diagnosis, especially if followed by signs of bronchial obstruction and pulmonary collapse of the same region. Pneumonia might, however, give these same signs and it is at times associated with fibrinous bronchitis. Consolidation would not disappear immediately on expectoration of a cast, as may occur with collapsed lung from obstruction of the tubes. The temperature is unsafe as a guide, as, during the acute symptoms, the temperature may be as high in severe cases of fibrinous bronchitis as it would be in pneumonia.

Chronic bronchitis is essentially different from pulmonary phthisis, with which it is most easily confused, in its effect upon the general condition of the subject. There is, in chronic bronchitis, more or less distension of the upper portions of the chest from emphysema. The clavicles are elevated, the accessory muscles of inspiration are prominent in the neck, as the vertical expansion of the chest is increased. The supraclavicular depression is increased and in emphysematous chests is filled, during coughing, with the distended subclavian and innominate veins. The nose and ears appear thickened, the conjunctivæ œdematous, the face turgid or suffused. The skin is dry, loose and non-elastic, and the veins are enlarged, tortuous and dark. All this is in distinct contrast to the chronic consumptive. The signs elicited by palpation and percussion depend on the relative amount of emphysema, peribronchitis and induration of the lung tissues which may have developed. Increased fremitus may be felt in the dilated portions of the chest (upper portion), provided the connective tissue is increased somewhat, and the air pressure is marked. The percussion note is hyperresonant but varying in character over the upper portion, and may be hard and

quite dull over the lower portions if these are indurated, well-blocked with mucus, or are collapsed, œdematous or congested. All varieties of dry and moist rales are heard. The expiration is lengthened and increased in pitch. The wavy respiration of bronchiectasis may be heard at times after a coughing spell. Syphilitic gummata, aneurisms and mediastinal growths may produce symptoms easily confounded with chronic bronchitis. Healed or quiescent cases of tuberculosis with emphysema and bronchial catarrh may easily be mistaken, particularly as these cases do not always exhibit bacilli in the sputum. Tuberculosis may be engrafted on chronic bronchitis and emphysema and not be recognized without examination of the sputum.

On the other hand we occasionally find a few bacilli in the sputum of chronic bronchitis merely as an accidental matter; hence, a few bacilli in the sputum is not always to be regarded as evidence of tuberculosis of the lungs. When, however, the physical signs are more marked in one or both apices, tuberculosis may be suspected whether bacilli are found in the sputum or not.

TREATMENT.—The remedies for bronchitis are legion, as the clinical index of any work on therapeutics will attest. Many of these remedies, while of undoubted value, are as certainly disagreeable and nauseating. A mixture of eight or ten ingredients is as unscientific as it is objectionable pharmaceutically. The simplest remedy is the best.

In *acute bronchitis* the natural course is towards recovery, and a little well directed aid will usually be sufficient. In the dry stage we wish to relieve pain, bronchial irritation, and promote secretion. If the pain be severe and the patient distressed, hot poultices, sinapisms, turpentine liniment or soap liniment with tincture of capsicum added may be of service. Dry cupping may be useful at times. For the soreness and irritation in the trachea and main bronchi, nothing is better than an oil spray, using benzoinol (plain), albolene, etc. The patient can readily be taught to inhale these sprays. The oil, besides protecting the membrane from the air, also induces secretion, which is the next object to be attained. For this purpose, ipecac, antimony, or tartar emetic are usually em-

ployed in doses not sufficient to cause emesis. In robust pa-
tients, the quickest way to induce secretion is to put one grain
of tartar emetic in a cup of water and give a teaspoonful
every ten minutes until there is slight nausea (Thomson).
The syrup of ipecac may be used in children for producing
secretion, in five drop doses every hour to a child two years
old. Small doses of Dover's powder (one grain) every hour
can be used in adults. Small doses of aconite frequently re-
peated are useful in children in the early stage to lessen fever
and circulatory excitement. When secretion has begun and
the cough is troublesome, as it usually is by the time the case
comes under observation, the indications are to loosen the
secretion so that it can be removed with as little cough as pos-
sible, and to quiet the irritability of the bronchial membrane,
and lessen the cough. For these purposes the following may
be used:

℞

         Am. mur.,   ℥ii.
         Codeine sulph.,   gr. v—vi.
         Liq. am. acetat.,   ℥i.
M.   Syr. tolu.,   q. s. ad ℥iii.
  S.   Teaspoonful once in 2 to 4 hours.

Syrup of yerba santa or glycyrrhiza may be used to better
cover the taste of the ammonia salt if advisable. When there
is the constant hacking cough or laryngeal irritation the follow-
ing more sedative combination may be used:

℞

         Am. mur.,   ℥iss.
         Morph. sulph.,   gr. iiss.
         Fl. ext. cannabis ind.,   gtt xx.
M.   Syr. tolu.,   q. s. ad ℥iii.
  S.   Teaspoonful every two hours.

When the acute symptoms are past, but the cough with
moderate secretion still persists, pills of terpin hydrate, or
syrup of hydriodic acid, or syrup of lactucarium may be of

service. At this stage, especially in old people, the following is useful:

R

Sodii iodidi,	ʒiss.
Codeine sulph.,	gr. v.
Fl. ext. grindel. robust.,	ʒvi.
M. Syr. tolu.,	q. s. ad ʒiii.
S. Teaspoonful thrice daily.	

This mixture is particularly effective in localized bronchitis. Opium should not be given to young children with bronchitis, unless in very small doses of such a preparation as the camphorated tincture. In localized bronchitis in children the syrup of hydriodic acid is very useful. In children with irritating cough and hoarseness, the following is of benefit:

R

Am. mur.,	gr. xlviii
Tr. anemones praetensis,	ʒi—ii
Liq. am. acetat.,	ʒvi.
M. Syr. tolu.,	q. s. ad ʒiii.
S. Teaspoonful 3 to 4 times daily.	

For persistent dyspnœa and lividity in bronchitis Reilly recommends thirty minims each of sp. ether and aromatic sp. of ammonia, twenty minims of tinc. aurant. am., with two drams of camphor water and enough distilled water to make one ounce. To be used when necessary.

In *bronchiolitis* we have a more serious condition to confront. In order to lessen the acute stage we may give aconite in sufficient doses (one or two drops in liquor of the acetate of ammonia every two hours). If any depression results a little brandy will remove it. In children, in the absence of lobular pneumonia, poultices are to be discountenanced, nor is opium admissible; it adds to the danger of pulmonary collapse and pneumonia from mucous obstruction of the weakened bronchial tubes. If the mucus is tenacious, a steam spray

from lime water may give relief. Two grains of muriate of ammonia, two or three drops of tincture of bryonia, and fifteen drops of the liquor of the acetate of ammonia, should be given every two hours. If the respiration is above thirty-five per minute and difficult, five drops of tincture of nux vomica should be given every two hours. In adults, from 1/30 to 1/20 of a grain of strychnia may be used.

When dyspnœa is severe and mucous rales show that mucus is obstructing the tubes, it is necessary to aid in expelling the secretion by causing vomiting. Ipecac, antimony, and apomorphia tend to increase secretion and the relief is not apt to be as permanent as when we use sulphate of zinc and alum (five grains of each), or yellow sulphate of mercury (two to five grains).

The chest should be covered by two or three layers of flannel or a wadding jacket. In the latter stages, stimulating applications to the chest, one or two drams of brandy every hour, small and frequent drinks of hot milk, may all be of service in general stimulation. The intimate association of bronchopneumonia with bronchiolitis, makes many of the means used in the former condition of interest here, but these will not now be considered.

In adults, stimulation with alcohol and strychnia is necessary in the late stages of severe cases. The diet for children should be milk with lime water. For adults milk and seltzer water is best.

Fibrinous bronchitis is not particularly amenable to treatment. During the attacks, the continuous inhalation of vapor of creosote, guaiacol, or alkaline substances, may be of use. Iodide of potassium in large doses is recommended and should be tried. Intra-tracheal injections of glycerine have been thought to aid separation of the casts. Olive oil or liquid vaseline may also be used. In the recurrent forms, change of climate may be tried, though the results are very uncertain.

Chronic bronchitis, of all bronchial inflammations, will tax the resources of the physician most. The tendency to relapse and the poor nutrition of the subjects of chronic bronchitis, are the chief factors in the obstinate course of the disease. Climatic treatment obviates, in a measure, the proba-

bility of relapse. Some cases do well in a warm, moist air, like the west Florida coast or the Bermudas. Others do best in a warm, dry climate, such as is found in the southwestern states, or the north-western states during summer. In all instances sunshine is the main requirement.

The nutrition is to be improved by nourishing and easily assimilated food and by tonic medicine. Iron is the chief remedy of this kind. The iodide of iron is particularly beneficial, though the tasteless tincture, citrate of iron and ammonia, and Basham's mixture are all eligible. Strychnia should be given to all elderly people with chronic bronchitis, both as a respiratory and cardiac stimulant. In general or localized chronic bronchitis in children the syrup of hydriodic acid is an efficient remedy. Cod-liver oil has long done duty in chronic bronchitis. To those who can take it it is a valuable remedy. The so-called alkaloids of cod-liver oil (morrhuine, gaduine, etc.) appear to exert as much influence for good, on chronic bronchitis, as the oil itself, and they are much more eligible for administration. A number of histories might be cited proving this point. In a child eight years old who suffered from bronchitis for five years, and who had received all kinds of treatment, including cod-liver oil, which she was fond of, complete recovery occurred in three months while under treatment with cod-liver oil alkaloids. The combinations of these substances with guaiacol (liquid or carbonate) or creosote, is of good service. These latter drugs are more beneficial in small doses than in large ones, and should not be given in doses which disturb the stomach in the slightest. Terebene is a valuable remedy, although somewhat nauseating. It is best given in emulsion made with acacia, in doses of ten to fifteen drops. It is well adapted to cases with scanty, viscid secretion.

For the morning cough, Burney Yeo advises sodium bicarbonate, gr. xv; sodium chloride, gr. v; spirits of chloroform, m. v, in anise water, taken with a little warm water before rising. In old people a mixture containing iodide of sodium or potassium (gr. v), tincture of belladonna (gtt. iii), fluid extract of grindelia robusta or fluid extract of euphorbia pilulifera (gtt. xv-xx), and sufficient quantity of syrup of tolu

or syrup of yerba santa, will be found very useful.   Iodide of
sodium and fluid extract of grindelia robusta are serviceable in
localized bronchitis in adults.   When cough is persistent from
$\frac{1}{4}$ to $\frac{1}{3}$ of a grain of phosphate of codeine every four hours
will be found of service.

One of the most efficacious means of treating various
forms of chronic inflammation of the upper bronchial tract is
by means of sprays.   The vapor from a cup of boiling water
containing a teaspoonful of compound tincture of benzoin is
very soothing.   Albolene or benzoinol may be used as a ve-
hicle for sprays.   In dry catarrh a 2 per cent. solution of
menthol, camphor, oil of eucalyptus, chloride of ammonium,
wine of ipecac, etc., may be used.   In dry asthmatic bronchitis,
lobelia, internally, is useful.

With bronchorrhœa we may use tar, turpentine, terebene,
copaiba, senega, terpin hydrate, or hydrastis canadensis.   In
fœtid bronchitis, sprays or inhalations of aristol, menthol or
creosote may be used.   In severe cases the creosote vapor bath
may be tried as recommended by Chaplin (*vide* Bronchiec-
tasis).

In gouty bronchitis, much advantage will be found in
colchicum in connection with iodides.   The various mineral
waters which are applicable to gouty conditions should be used.
Much good will result from an occasional mercurial cholagogue
followed by some aperient water.   In syphilitic bronchitis
iodides and mercury are, of course, the chief remedies.

In the later stages of chronic bronchitis in old people,
when the right heart becomes dilated and dyspnœa is severe,
oxygen gas, large doses of strychnia hypodermatically, and,
under some conditions, venesection may be indicated.   When
the expectoration is sticky and difficult to remove from the
mouth and throat, these passages can be kept clean and free
from mucus by rinsing the mouth with a little peroxide of
hydrogen.

BRONCHIECTASIS.

Dilatation of the bronchi is comparatively rarely observed
clinically.   Anatomically it is not uncommon in its association

with tuberculosis and other morbid processes of the lungs and pleura. In adults it is usually a secondary, chronic affection. In children an acute form occurs, usually in association with secondary bronchitis in rickety children (Carr). Bronchiectasis is most common in males. It occurs at any age, but is most common during middle life.

ÆTIOLOGY.—Bronchiectasis is always secondary to some disease of the bronchi, lung or pleura. Chronic bronchitis, pulmonary cirrhosis, broncho-pneumonia, pulmonary tuberculosis and chronic pleurisy are its most frequent associations. Changes in the bronchial wall and peribronchial tissue which weaken and destroy the elasticity of the bronchi, are the immediate factors in the production of the dilatation. These changes are most often induced by chronic bronchitis, stenosis or obstruction of the bronchi, pulmonary collapse, pneumonia, emphysema, cirrhosis of the lung, and the various effects of chronic pleurisy.

MORBID ANATOMY.—The dilatation of the tubes may be either cylindrical or saccular. The dilatations may be connected by tubes of normal calibre—the moniliform dilatation of Cruveilhier. Cylindrical dilatation occurs most often in the larger tubes. Usually there are constrictions at various points. Saccular dilatation may involve large sections of the lungs, the lung tissue having disappeared and in its place are numerous sacks with an opening in the bottom of each. A whole lobe may be thus affected ("turtle lung"). The terminal bronchi are usually affected first and the size of the dilatation is proportionately greater than in the larger tubes (Biermer). The terminal extremities of the tubes are closed or obliterated early in the disease. In a large proportion of cass both lungs are affected. The acute form in children involves many tubes. When dilatation results from obstruction of a tube, collapse of lung tissue, pleurisy, etc., it is limited to the area of lung involved. The lower lobes are most often involved primarily, the apex when secondary to tuberculosis.

The bronchial tubes exhibit the various evidences of chronic inflammation of the mucosa. The walls and peribronchial tissue are thickened. In the sacculated form the walls may be thin. The tubes may be empty or may contain

purulent, fœtid fluid of a gray or yellowish color, curdy or inspissated; or, they may be filled with a clear, jelly-like mucus without any particular odor. Various changes occur in the lungs and pleuræ. Areas of gangrene occur around dilated tubes. In the acute form in children the chief changes are: Acute peribronchitis; the presence of innumerable small cavities; small air-containing vessels on the surface of the lungs (Fowler).

CLINICAL HISTORY.—Paroxysmal cough with the expectoration of quantities of purulent and offensive secretion, more or less dyspnœa, some pain, occasional hæmoptysis and occasionally fever, are the usual features of the history. The cough occurs in the morning or evening. As a rule the intervals are comparatively free from cough and expectoration. The quantity expectorated at once, varies from two to ten or twelve ounces, and from half a pint to two quarts in twenty-four hours. If drainage is good the cavity will empty completely at a paroxysm of coughing. Some cavities only drain when the patient is in certain positions. The odor of the sputum or breath may be very offensive or it may not be noticeable.

The following case was one of saccular dilatation of the lower branch of the left bronchus:

Young woman aged 22. Had a prolonged attack of bronchitis when seventeen years old. Has had paroxysmal cough ever since with considerable expectoration. For last four years has had coughing spells in morning or evening, when she would expectorate a quantity of mucus. For last two years these spells occur in the morning shortly after rising. By getting in the knee-chest position with the left shoulder as low as possible she is able to raise about six or eight ounces of sputum in a few minutes, and is then free from cough for the most of the day. The sputum is seldom offensive. The expansion of the left side is slow and somewhat less than of the right. The fremitus is slightly increased below the level of the third rib, percussion note nearly normal, inspiration interrupted and wavy over a space extending from the third to the fifth ribs in the anterior axillary line. Loud metallic rales are heard over the same area.

SYMPTOMS AND DIAGNOSIS.—The physical signs obtained in cases of bronchiectasis show every possible variation in accordance with the conditions with which it is associated. The percussion note may be dull if the cavity is full; if empty, the percussion note is raised, hollow or extra-resonant according as the fibrosis is marked or not. Cracked-pot sound may be obtained in tubercular dilatations of the upper lobe. In cylindrical dilatation the respiratory sound will be tubular, blowing or hollow. Inspiration and expiration may be wavy in character. In saccular dilatation the breathing may be cavernous. There may be loud, gurgling rales. At the end of inspiration a short puff or blow may be heard, seemingly close to the ear (the "veiled puff" of Skoda). The wavy, irregular character of the inspiration or expiration, and loud metallic crackles or gurgles over a limited area, are the most characteristic evidences of bronchiectasis. When a whole lung is full of small cavities the percussion note may be tympanitic and the auscultatory signs will be ill defined and low in pitch. Clubbing of the fingers and toes may be marked.

The character of the sputum and the method of expectoration generally suffices to distinguish bronchiectasis from emphysema. Pulmonary gangrene is generally secondary to acute pneumonia or to tuberculosis. Examination of the sputum is generally necessary to exclude tuberculosis, though the course of the two affections is different. Tuberculosis may occur secondary to bronchiectasis. Localized empyema opening into the lung may be confused with bronchiectasis, but its history, associated conditions of the lung and pleura, and puncture, when that is possible, will differentiate.

Bronchiectasis is rarely, if ever, recovered from. It may end in gangrene, lobular pneumonia, fatal hæmoptysis, or some cardiac or renal complication.

TREATMENT.—The indications are to maintain the general strength and nutrition and to disinfect the bronchial tract. Superficially situated dilatations, especially of the larger tubes, may be disinfected by inhalations of aristol, menthol, eucalyptus, creosote, guaiacol, tar or turpentine sprays.

,In a woman of 35, who had suffered from a bronchial dilatation in the anterior middle portion of the right lung, and who expectorated over a pint of mucus every twenty-four hours, disinfection of the expectoration, which was very offensive at times, was accomplished by the inhalation of a spray of aristol (3 per cent.). This was used every other day and made the trouble quite bearable.

More energetic treatment consists in the continuous inhalation of coal-tar, creosote, guaiacol or cresoline vapor; intratracheal injections; and hypodermic injections of sterilized guaiacol or creosote. The vapor bath (Chaplin) is given in a small room or compartment clear of furniture. Commercial creosote is heated in a metal saucer over a spirit lamp until clouds of vapor fill the room. The patient's clothes are protected by a cover, the eyes by goggles, the nares by cotton plugs, and the hair, in women, by a bag. The bath is given every other day at first, for fifteen or twenty minutes, gradually increasing to an hour every day. The bath produces violent coughing and profuse expectoration and sometimes vomiting.

Intratracheal injections (Rosenberg) are made by injecting a dram of the solution into the trachea by means of a syringe, passing the nozzle of the syringe below the vocal chords. Menthol (5-10 per cent.), guaiacol (2 per cent.), singly or combined, in olive oil, is recommended. By the hypodermic method, thirty minims of a twenty-five per cent. solution of guaiacol or creosote in sterilized olive oil, may be used but does not appear to have any advantage over the inhalation methods, of which the vapor bath is. according to Fowler, decidedly efficacious.

Surgical treatment of a bronchiectatic cavity is seldom possible, though in some instances may be advisable.

BRONCHIAL STENOSIS.

General narrowing of the bronchial tubes resulting from inflammatory swelling, catarrhal, croupous or diphtheritic exudation is not included under this caption.

ÆTIOLOGY.—Local bronchial stenosis arises from foreign

bodies in the bronchi, cicatricial contraction of bronchial ulcers, sclerosis of the bronchial wall, malignant growths, aneurisms, tumors of the lung or mediastinum or hydatid tumors.

MORBID ANATOMY.—Foreign bodies are apt to lodge in the right bronchus as that branch is more nearly in line with the trachea in most individuals. Articles several inches in length may, however, enter the left bronchus. Cicatricial stenosis are almost invariably of syphilitic origin. Gummatous infiltrations of the mucosa break down, ulcerations result and the subsequent scars and thickened sub-mucous layer cause stenosis in one or several situations. In chronic disseminated tuberculosis there may be stenosis of the tubes from thickening of the wall at points adjacent to a fibrous tubercular nodule, and also from the cicatricial wall which surrounds a cavity, involving also the entering bronchus (Powell). A stenosis effected gradually, tends to the production and retention of secretion behind the stenosis and results in dilatation of the tube and fibrosis of the surrounding tissue.

A cavity may become closed and its contents become cretaceous, or there may be intermittent discharge of its contents under pressure of accumulated secretions. In rapidly developed and complete stenosis there is collapse of the area of lung tissue supplied.

CLINICAL HISTORY.—The history is variable. It is astonishing what tolerance there is for foreign bodies in the bronchi, provided they do not lodge at any of the specially sensitive points.

A boy 15 years old, placed a blow pipe over a hat pin which was loosely stuck in the floor. (The pin was steel, about six or seven inches long and with a round ebony head.) In attempting to suck the pin out of the floor he drew it into his throat. He had a short coughing spell which soon subsided. The pin had disappeared. During the next day there was occasional coughing spells which were not severe. Examination showed slow expansion of the left lung, partial suppression of respiratory sounds in the same side with exaggeration of the respiration on the right side. There were no local signs of obstruction. Tracheotomy was performed and the

pin found in the left bronchus. It was broken in the effort of extraction, the head and about three inches of the shank being obtained. Three days afterward the remainder of the pin was coughed up. No subsequent ill effects.

Stenosis of the main bronchus will cause dyspnœa and there is more or less cough and expectoration which may be similar to that of bronchiectasis. The cough may be spasmodic or laryngeal in type. There may be pain if pulmonary collapse and dry pleurisy occurs. Tumors, aneurisms and syphilis will modify the history.

SYMPTOMS AND DIAGNOSIS.—Partial stenosis gives rise to diminished expansion of the affected side, recession of the interspaces, costal margin, episternal and supraclavicular regions; diminished vocal fremitus, resonance and respiratory murmur. Rales and fremitus may be obtained over the site of the obstruction, and interscapular stridor may be heard. When the stenosis is of long standing or is complete, signs of bronchiectasis, pulmonary fibrosis, consolidation or collapse, with contraction of the side, will appear. The unaffected lung shows a greater or less degree of compensatory action with a corresponding intensification of the physical signs.

When the examination leaves doubt as to the nature of the obstruction, the probabilities favor aneurism, mediastinal growth, or cicatricial contraction from syphilitic gumma (Fowler). In stenosis from aneurism and malignant growths the signs of these conditions develop early. Stenosis in association with consolidation and profuse expectoration may be mistaken for tuberculosis, empyema, or pneumonia. The diagnosis is sometimes very difficult.

TREATMENT.—The treatment is largely symptomatic. In syphilitic or aneurismal stenoses, iodide of potassium in large doses should be used. Chloroform or oxygen may be used for the paroxysmal dyspnœa caused by aneurism. Powell recommends nitroglycerine for the same purpose. Expectorants may give some relief, though as a rule they are of little use. In bronchial stenosis from foreign bodies we should resort to surgical measures whenever there is the least possible

hope of relief by such means. A young man, recently under treatment, had a vertebra of a chicken lodged in his right bronchus. He developed hectic fever, emaciation, and expectorated purulent material. Resection of the fourth rib anteriorly with exploration of the lung failed to dislodge the bone. Subsequently he developed collapse of the lower posterior portion of the right lung. Resection of the ninth rib posteriorly was performed, the posterior bronchus was opened, air entered freely, and the opening was plugged. The next morning the patient had a severe coughing spell and coughed up the bone. He made a good recovery, and beyond some limitation of the action of the right lung and some cough, is at present in good health.

CHAPTER IX.

ASTHMA.

For clinical convenience and from an ætiological basis, several varieties of asthma have been recognized. It will be found that these different forms of the disease represent simply symptomatic variations of an affection which is neurotic in nature, spasmodic in occurrence, and is characterized by a disturbed innervation of the bronchi which manifests itself in spasm (Liebert) of the bronchial muscles and is associated with spasm of the diaphragm and correlated respiratory muscular apparatus, resulting in a special type of dyspnœa.

Asthma may occur at any age. According to Salter's statistics, thirty-one per cent. of the cases occurred during the first decade of life. It is generally stated that asthma is twice as frequent in males as in females, though if we include only the cases of typical spasmodic asthma, it is evident that it occurs as frequently in the one sex as in the other.

The most individual clinical forms of asthma are the so-called *idiopathic* or true *spasmodic* asthma, *bronchial* asthma, *dyspeptic, toxic, cardiac* and *hay* asthma. These terms serve only to specify certain ætiological factors which are associated with dyspnœa of asthmatic origin.

ÆTIOLOGY.—Thirty-five per cent. of asthmatics give a history of heredity, most often from the paternal side (Thomson). Asthma is generally associated with a family tendency to bronchitis or other pulmonary disorders or to nervous affections. There may be a predisposition existing through one generation only, or a distinct history of heredity. Bronchitis (thirty-seven per cent.—Salter), fibroid tuberculosis, epilepsy and various nervous disorders are frequently associated with asthma.

The exciting causes of the asthmatic paroxysm may be of central or peripheral origin. The central causes may be emo-

tional or neurasthenic or may be due to toxic irritations from drug poisons, gout, diabetic or nephritic toxæmias (*uraemic asthma*) or to intestinal toxæmia (the so-called *humoral* form of *peptic asthma*). Among the neurasthenic cases are those induced by fright, worry, the excitement of important social duties or the nocturnal cases which occur with darkness and can be obviated by keeping a light in the sleeping-room.

The asthmatic attacks which occur directly after the ingestion of certain kinds of food and are accompanied by gastric disturbance, are probably of peripheral origin, and according to Salter are due to bronchial contraction produced through reflex irritation of the pulmonary filaments of the pneumo-gastric from irritation of the gastric terminations of the same nerve. The most frequent seat of peripheral irritation resulting in asthma is the bronchial tract. Climate, locality, moisture, dust, etc., are factors which are closely connected with bronchial susceptibility in the production of an asthmatic attack, but even here nervous conditions seem, at times, to be potent in producing the prodromal bronchial symptoms which precede the asthmatic attack, as appeared in the following case :

Farmer, 39 years of age. Lived in a dry, healthy part of Iowa. For last ten years has suffered from asthma whenever he visited any of the large cities. The attacks are preceded by bronchial symptoms. At home, on his own farm, is never troubled. Last year he built a new house three-quarters of a mile from his old residence, but was unable to occupy it because of severe asthmatic attacks which appeared whenever he remained about the new house for a day at a time. The attack would be preceded by cough, wheezing and expectoration, beginning within two hours after he reached his house and resulting in a paroxysm of asthma within eight or ten hours. When he returned to his old residence the attack would cease within an hour or two. The attack never appeared while the new house was in process of construction, but only when he attempted to live there, and at the same time he could visit with impunity a new barn in process of building on his old farm.

Laughing, coughing, sneezing, forcible breathing, etc., induce asthma by over-stimulating the bronchial muscles (Wilson Fox). Gastro-intestinal irritations are next to bronchial disturbances as a cause for asthmatic attacks. Very great variety and peculiarity is exhibited in relation to the various articles of diet which induce the attacks. Intestinal indigestion undoubtedly acts as a peripheral cause for asthma, but must also be classed among the causes of toxic asthma. Constipation and flatulence may also induce attacks of asthma. Nasal irritation acting through the sensory branches of the fifth nerve is a frequent element in peripheral irritations resulting in asthma. Nasal polypi, exostoses of the septum, turgescence of the cavernous tissue covering the turbinates and a thickening of this tissue, are given as a cause for asthma (Hack and others), and removal of this tissue has been insisted on by many. Undoubtedly many cases are due to nasal irritation and are relieved by its removal, but that mere turgescence of the tissues will induce asthma is not borne out by the clinical history of this very common condition of the nasal membrane, or by its removal in instance where it is the only nasal abnormality associated with asthmatic attacks.

Certain odors of animal or vegetable origin, dust, the pollen of flowers, the odor of certain drugs, may all induce asthma through the very erratic sensibility of some individuals to the irritation produced by these various substances. Unknown atmospheric conditions at certain seasons of the year and in certain localities induce asthma (hay asthma). Dental irritation in children may produce asthma. Uterine irritation from polypi, irritation of the pneumogastric nerve by neuroma or enlarged glands in the posterior mediastinum, vertebral exostoses or disease of the pneumogastric center may bring about attacks of asthma. Cold to the surface of the body, especially to the feet, may induce an attack, or it may be associated with the disappearance of a cutaneous eruption. Eczema, psoriasis and urticaria (Sir Andrew Clark) are frequently associated with asthma, the latter especially, as it is essentially a neurotic affection.

MORBID ANATOMY.—It is useless to review the various theories regarding the exact condition of which the paroxysm

is the result. The nervous factor is generally recognized,—some condition of the nervous system by which stimuli, ordinarily of no moment, influence the vagus or sympathetic nerves. Whether the motor branches of the vagus are alone concerned (spasm of the bronchial muscles) or whether hyperæmia of the bronchial mucous membrane (Weber) from local vaso-motor paresis of sympathetic origin is also a factor, is a disputed question. The occurrence of both of these conditions most fully explains the various types of the disease. Bronchial spasm (Williams and others) is a main factor in the attacks, but some degree of spasm of the diaphragm (Wintrich, Bamberger) and possibly of the inspiratory muscles (Jaccoud, Sée) is, in some instances, necessary to explain the clinical manifestations.

There are no characteristic lesions of asthma. Various changes in the bronchi and pulmonary tissue may occur, and, in severe and prolonged cases, dilatation of the right side of the heart will develop. The effect of the bronchial obstruction is to cause increased inspiratory effort with still greater difficulty in expiration; as a result there is a gradual increase in the amount of residual air in the lungs and over-distention of the alveoli.

The amount of expectoration in asthmatic attacks is very variable. In the bronchial type it may be abundant. Generally, towards the end of the paroxysm, expectoration is more or less free and consists of semi-transparent, grayish mucus in pellets —"boiled tapioca" expectoration. Spiral corkscrew threads of mucin (Curshman's spirals) and pointed octahedral crystals (Charcot—Leyden crystals) are found in the sputum. The latter are supposed to be a combination of phosphoric acid with an organic base. While spirals and crystals are not found as frequently in other pulmonary affections as in asthma, they are in no way characteristic of the latter affection.

CLINICAL HISTORY.—Very great variation is shown in the history of the different types of asthma. When developed in childhood it may disappear in time. The longer the duration and the more frequent the attacks, the greater will be the degree of associated emphysema and the less likely the probability of recovery. There may be a history of various peculiar

and interesting exciting causes or there may be no exciting cause ascertainable. There is a wide range of associated diseases and morbid states, or there may be absolutely no local or general condition discoverable even in the most severe attacks of the true spasmodic form of the disease.

The attacks may occur only at certain seasons of the year. They may be weekly, monthly or yearly, or be separated by several years. Periodicity is a marked feature of nearly all forms of asthma. The spasmodic type is apt to come on at night after a few hours sleep. There is apt to be a feeling of somnolence or depression preceding the attack, with or without bronchial symptoms. In bronchial asthma there is every degree of combination of bronchial and asthmatic symptoms. The dyspnœa may be more or less continuous and the asthmatic feature merely an exacerbation when some exertion overtaxes the weakened heart and distended lungs. Many of these cases, while termed asthmatic, will hardly come under that designation. The ætiological relations of asthma give many interesting points in the clinical history of the various types of the disease which are not necessary to recount.

SYMPTOMS AND DIAGNOSIS.—In the markedly spasmodic forms of asthma the attacks may come on suddenly and without warning, especially at night, though there are generally some premonitory symptoms, such as depression or restlessness, wheezy breathing, abundant, pale, limpid urine, constriction about the chest and some dry cough. The subject may be wakened from sleep by difficult breathing, which soon becomes severe dyspnœa. He sits up, leans forward, breathes slowly and laboriously. The face is slightly congested or pale, there is profuse perspiration and an anxious, careworn expression. The shoulders are rounded and elevated and the hands grasp a chair or table in order to fix the scapular and humeral attachments of the chest muscles. The jugular veins are distended and the muscles of the neck are prominent. The whole chest is lifted during inspiration, which is, nevertheless, short and inefficient. Expiration is three or four times its usual length, but is more difficult and inefficient than inspiration, and the chest becomes more or less fixed in a condition of inspiratory distension. There is no pause after expiration, inspiration begin-

ning immediately, suddenly and forcibly. The respiratory rate may not be increased; the pulse is rapid and feeble and may disappear during inspiration (*pulsus paradoxus*); the temperature is normal or depressed; and the patient, while in the severest distress, is not alarmed. Persistent itching of the chin, sternum, or between the scapulæ may be present during the earlier part of the paroxysm (Salter).

The thorax is much distended; the ordinary clothing may not meet around the chest by two inches. The diaphragm is lowered as much as possible, the supra-clavicular fossæ recede during inspiration and there is absence of the usual areas of cardiac and hepatic dullness with epigastric pulsation. The percussion note is hyper-resonant, vocal fremitus is absent, the vesicular murmur is feeble or absent, or is covered by high-pitched sibilant rales and sonorous rhonchi which are very changeable as to location. If bronchitis be present the rales may be more or less liquid or crackling in character. The long, wheezing expiration and the accompanying dry rales are positive proof of bronchial spasm. Every possible gradation as to severity and duration of attack is met with in the true spasmodic form of asthma, as well as various degrees of bronchial symptoms which remain after the attack in the bronchitic form. After an attack of purely spasmodic asthma has subsided there may not be a single abnormal sign about the patient's chest. In severe cases cyanosis may be marked and capillary retinal hæmorrhages may occur (Walsh). The urine is abundant, pale and of low specific gravity and the solids are diminished (Ringer).

The dyspnœa of asthma is distinguished from that of laryngeal or tracheal stenosis by its expiratory, instead of inspiratory, nature and by wheezing instead of stridor. Laryngeal dyspnœa is accompanied by increased movements of the larynx and by diminution in the size of the chest and elevation of the diaphragm. In cardiac asthma there is rapid, panting or sighing breathing and the expiratory act is not prolonged. In aneurismal dyspnœa there is tracheal tugging, localized dullness, pulsation, and brassy cough as concomitant signs, with, perhaps, signs of pressure on the bronchus,—diminished breathing in the left upper lobe, aphonia from vocal paralysis.

TREATMENT.—Asthma rivals bronchitis in the number of medicaments employed for its relief. This is owing to the neurotic nature of the disease and the consequent varying ætiological factors. These factors must be carefully studied in the individual case and the treatment directed accordingly. The patient's knowledge and observation of his own case is often most useful in directing the treatment.

Relief of the paroxysm in no wise influences the continuance of the disease. The remedies used for the relief of the paroxysm are all neurotics which have only temporary symptomatic effects, and if the disease is to be cured it must be done through the administration of constitutional remedies, such as iodides, arsenic, iron, strychnia, quinia, cod liver oil, etc. These remedies are to be persisted in for long periods and are much aided by controlling the attacks in the meantime by the neurotics. Iodide of sodium, strychnia and fluid extract of euphorbia pilulifera is a combination whose prolonged administration has given me much satisfaction, especially in the bronchitic form of asthma.

Climate is an important factor in the treatment of the asthmatic. Here each case is a law unto itself, very striking peculiarities being observed in relation to climate. Generally speaking, a moderately elevated, dry climate is best, particularly in the pine or fir regions. It is generally stated that the smoky, gas-laden air of large towns is agreeable to asthmatics. This may be true of the purely spasmodic form of asthma, but in my experience is not so of the bronchitic form, in which such cities as Pittsburg, in spite of its comparatively favorable latitude, elevation and location in the petroleum belt, is entirely unsatisfactory as a residence for asthmatics with bronchial symptoms. Chicago is to a like extent objectionable because of the rapid and radical changes in temperature and moisture. Northern New York, Wisconsin, Michigan, Minnesota, Idaho, Wyoming, etc., are particularly favorable localities for the asthmatic. In the spasmodic form due to susceptibility to atmospheric conditions incident to certain seasons of the year, and in hay asthma, a sojourn at the seaside is often beneficial. Many of these cases, especially of hay asthma, are entirely free from attacks while at Mackinac Island, a locality

that, while entirely surrounded by water, has a particularly dry, exhilarating atmosphere. Cases of asthma which have developed marked emphysema must not be sent to a high altitude because of the resulting strain on the right side of the heart.

In preventing attacks of asthma each case must be treated symptomatically. Peptic asthma involves the treatment of every variety of dyspepsia. In addition to dieting, the administration of twenty grains of strontium bromide and ten grains of subcarbonate of bismuth, three or four times daily, is of great benefit. Flatulence, constipation, uterine irritation and nasal irritation must be relieve. In the cases exhibiting great nasal sensibility to odors, gases, temperature, etc., with hyperæmia or turgescence of the turbinate coverings, it is best to treat them persistently with soothing, cleansing and disinfecting sprays or steam inhalations. In the absence of abnormal bony or soft tissue growths or septum deviations, operations for the removal of tissue or cauterizations are not advisable. The ultimate results are not nearly as good as many rhinologists have inferred from the immediate effects of such treatment. For nocturnal attacks of spasmodic asthma, thirty grains of potassium bromide and a dram of Hoffman's anodyne, at bed time, will often prevent attacks (Thomson). Iodide of sodium or potassium and arsenic are the most useful prophylactic drugs. Tincture of lobelia and fluid extract of stramonium are useful adjuvants. The iodides should be gradually increased until a fairly large daily dose is given without producing physiological effects. Hot coffee, a liberal drink of whisky, the fumes of burning nitre paper (blotting paper soaked in a solution of nitrate of potash, gr. xxx ad ℥i) or tobacco will at times ward off an attack of asthma. Skerret recommends 5 to 10 grains of citrate of caffein at bed time for the prevention of morning attacks, and five grains every four hours during attack in bronchial asthma. The vapor of turpentine or ammonia, or painting the pharyngeal wall with a solution of ammonia (Durcos, Trousseau) or swabbing the nasal passages with a solution of cocaine, may abort attacks in cases of nasal and pharyngeal irritation.

In bronchial asthma, besides the palliative treatment, the following is useful:

℞

Natrii iodidi,	℥ii-iii
Tr. nucis vom.,	℥iv
Tr. belladonnæ,	℥i-ii
Spts. eth. sulp. co.,	℥iss
Elix. q. s. ad	℥vi

M.

Sig. ℥ii every 6 hours.

Jackson recommends the following mixture:

℞

Sodii iodidi,	gr. ij
Sodii bromidi,	gr. ij
Ext. euphorbiæ pil. fl.,	m. iij
Tr. lobeliæ æther.,	m. iij
Nitroglycerini,	1-200 gr.
Aquæ,	ad fl. ℥ss

M. Sig. One tablespoonful at a dose.

For the relief of the paroxysm of asthma we rely almost entirely on the neurotics and stimulants. Belladonna, hyoscyamus, stramonium, duboisia, the nitrites (amyl, nitroglycerine, sodium), iodide of ethyl, opium and ether are the most serviceable. Much relief is obtained from the fumes of the numerous asthmatic cures, all of which contain more or less of the first four drugs enumerated. Candles designed for the administration of stramonium by inhalation (candelæ stramonii) are made of 150 parts of pulverized stramonium leaves, 70 of potassium nitrate, and three of balsam of Peru. Stramonium cigarettes (cigarettæ stramonii) contain one gramme each of stramonium leaves. Compound stramonium cigarettes (cigarettæ stramonii compositæ) contain one gramme each of a mixture of 3 grammes of stramonium leaves and 1 gramme each of the leaves of belladonna and tussilago farfara. The following is a good formula for a powder for asthma:

R

Belladonnæ,	fol.,	
Stramonii,	fol.,	aa ℥i.
Lobeliæ,	fol.,	
Silphii lacin.,	fol.,	
Potasii nitratis,	℥ss.	

M.   Fiat pulv.

Jackson's inhaling powder is the following:

R

Pulv. potasii nitratis,	ʒiv-ʒvi
Pulv. stramonii,	gr. lxxv
Pulv. lobeliæ,	gr. xc
Pulv. belladonnæ,	gr. xlv
Pulv. grindeliæ,	gr. xc
Pulv. hydrast. can.,	gr. xv

M.

Sig.   One heaped teaspoonful to be burned in a small, closed room or tent and the fumes inhaled.

Thomson recommends full doses of tincture of belladonna or an injection of atropine at the nape of the neck to arrest the asthmatic paroxysm. Nitroglycerine and chloroform are useful for an attack but the latter is more or less dangerous. A few drops of pyridine inhaled from a handkerchief or butter plate is often efficacious. Bruck has used ether subcutaneously with success. Riegel obtains prompt relief by the hypodermic use of from 0.3 to 1 mg. of atropine.

The most efficient remedy for an attack of spasmodic asthma is a hypodermic injection of morphine (1-6 gr.). The combination with atropine is advisable. There is little danger of drug habit and the best relief is obtained in the purely spasmodic cases. Morphia should not be given in cases of bronchial inflammation with dyspnœa and only in small doses, if at all, to cases of bronchitic asthma with pronounced bronchial symptoms.

The general management of the asthmatic involves much symptomatic treatment not necessary to detail here, but the more closely the individual case is studied the better will be the results.

PULMONARY EMPHYSEMA.

In pulmonary emphysema there is dilatation of the alveoli of the lungs and atrophy of the alveolar walls. Two forms are usually described; a vesicular, and an interlobular or interstitial form. The latter is entirely distinct from the former to which reference is always made by the unqualified term emphysema.

There are four clinical varieties of emphysema, which, while not constituting distinct forms of the disease, are yet sufficiently well marked to merit clinical division. They are *general emphysema* (volumen pulmonum auctum, chronic hypertrophic emphysema, large lunged emphysema,—Jenner), *senile emphysema* (small lunged emphysema, atrophic emphysema, senile atrophy of the lungs), *localized emphysema* (compensatory emphysema) and *acute emphysema* (dilatation of the lungs). Pathologically the last variety is not a form of emphysema, as there is no atrophy of the alveolar walls but simply acute dilation of the alveoli which may or may not be recovered from.

Emphysema may occur at any age, but is most common during adult life. It is more frequent in men because of greater exposure to predisposing conditions.

ÆTIOLOGY.—The distention of the air spaces is the primary change and precedes the atrophy of the alveolar walls. In the atrophic form it is possible that this order may be reversed. Exclusive of compensatory emphysema it is probable that failure of nutrition (Jackson, Waters) is a frequent element predisposing to the development of emphysema.

The increased air tension which dilates the spaces is brought about by increased expiratory force (Mendelssohn, Jenner) which first dilates those portions of the lungs which are in contact with the most yielding portion of the chest walls, i. e., the apices, anterior border of the upper lobes and the margins of the base of the lungs. The inspiratory theory (Laennec, Gairdner, Rindfleisch) is not now accepted as the general cause of emphysema, though in the production of localized emphysema the inspiratory cause is undoubtedly an important factor.

Bronchial obstruction, cough and muscular effort are the most important factors in producing emphysema. Occupations involving straining, lifting, playing on wind instruments tend to produce the disease. Chronic bronchitis, asthma, whooping-cough, laryngeal, tracheal and bronchial stenoses cause general emphysema. Localized emphysema may result from any disease of the lungs or pleuræ which limits the action of a portion or the whole of a lung. Compensatory emphysema of an entire lobe or lung may thus develop, or localized emphysema may develop in the lobules adjacent to areas of pulmonary atelectasis, infarctions or consolidation.

In those cases where emphysema is not accompanied by, or precedes the development of, those mechanical causes already enumerated, the cause rests in nutritive changes in the lung texture which destroys the elasticity of the lung and renders it unable to withstand the ordinary air pressure incident to the process of respiration.

Interlobular emphysema occurs from wounds of the lung, rupture of the alveoli from overstrain as in coughing, in connection with laryngeal diphtheria especially after tracheotomy. or in advanced cases of general emphysema from the rupture of air sacs.

MORBID ANATOMY.—The distention of the air passages begins in the inter-alveolar spaces (Rindfleisch). This increase is at the expense of the alveoli opening into the infundibular cavities in relation to which the alveoli are normally about one-third smaller. The dilatation may be primarily in the alveoli. The pressure diminishes the blood supply to the alveolar walls and their epithelium undergoes fatty degeneration. The septa between the alveoli become partially or completely destroyed and the walls between adjacent infundibular or alveolar spaces give way, the spaces coalesce and form round cavities of varying size which may be traversed by remnants of vessels or wall. There may be more or less connective tissue development, especially in connection with cases of chronic bronchitis.

These processes involve considerable destruction of the pulmonary capillary circulation. According to Rindfleisch communication is established between the pulmonary and the

bronchial veins which relieves the tension in the pulmonary artery but does not aerate the blood. The over distended lungs lose their elasticity and the inspiratory traction force exerted on the circulation is lost. This, together with the obliteration of the capillary vessels, increases the pressure in the venous system and the right heart suffers. Pressure in the vessels of the bronchial wall causes congestion of the bronchial membrane. The bronchi may be dilated, particularly in localized emphysema, or they may be obliterated or run as fibrous cords across the emphysematous sacs. The bronchial walls are usually thickened, though in atrophic emphysema they may be thinned.

The lungs are maintained in the position of ordinary inspiration. The diaphragm is lowered and the heart displaced downward and becomes more nearly horizontal. The hypertrophy of the heart is, in time, followed by dilatation, tricuspid regurgitation, dilatation of the right auricle and the usual congestions of the liver, stomach, kidneys, brain, and œdema of the tissues.

When interlobular emphysema occurs after tracheotomy, the air passes behind the deep cervical fascia into the chest. From the mediastinum it may pass along the tissue around the bronchi and blood vessels and appear on the surface of the lung as small beads or bubbles of air beneath the visceral pleura. Interlobular emphysema from any cause shows itself as small air bubbles in the interstitial tissue of the lung and in rows beneath the pleura. Pneumothorax and pulmonary collapse are the most frequent associated lesions of interlobular emphysema. There may also be general subcutaneous emphysema of the neck, face and arms or trunk.

In *general emphysema* when the chest is opened the lungs do not collapse. The margins are in contact with the sternum and the apices fill the suprascapular spaces. The auricular process of the left upper lobe occupies the præcordial space, the diaphragm is depressed, the lung tissue is soft, of a grayish color, somewhat pigmented, non-crepitant and may pit on pressure. The surface may have a frothy appearance or large rounded blebs may occupy the margins of the upper lobes, or the bases. These air sacs may be pedunculated. The lung

tissue is dry and bloodless except at the base where it may be œdematous or congested if bronchitis or weak heart have been present. Pleural adhesions are not so frequent as in other diseases of the lungs. Atheroma of the pulmonary artery is frequently present.

In *senile emphysema* the lungs are small and collapse readily and are deeply pigmented. The vesicles are fused from atrophy of the septa. The margins may be much dilated and large air sacs may be present. The bronchi are dilated and their walls thin, membranes congested and they may contain puriform fluid. Collapse and œdema of the posterior portions of the lower lobes are frequent. The chest is rigid and small and the lower ribs oblique and in contact with each other. Senile emphysema is associated with general atrophy and wasting of the bodily tissues.

Local emphysema is secondary to some disease of the lungs, bronchi or pleuræ, areas of arrested pulmonary tuberculosis, cavities, bronchial dilatations, atelectasis or fibroid areas in the lung or pleuritic adhesions and indurations. The apices and the posterior and upper portions of the lower lobes are frequent locations for this form of emphysema. Local areas of fibroid or collapsed lung may be surrounded by, or imbedded in, emphysematous tissue. Large air cavities are usually absent in compensatory emphysema.

The so-called *acute vesicular emphysema* is simply an over-distention of the alveoli. It may result from acute bronchitis, violent inspiratory efforts, partial stenosis of the trachea or bronchi by collapse of the lung increasing the strain on the remaining portion, asphyxia from any cause. While it occurs most often when the above causes are fatal, it also occurs in non-fatal cases and may be recovered from.

CLINICAL HISTORY.—The history of emphysema is in nowise characteristic. The insidious advent of the disease accustoms the subject to the altered conditions of respiration and there is little complaint until the condition becomes severe. The history mainly refers to the symptoms caused by the associated affections, particularly the bronchitis, and it is quite common to have patients who are markedly dyspnœic remark that if the cough were better they would get along all right

with the breathing. The dyspnœa is always present, is likely to be paroxysmal and is increased by exertion or interference with the action of the diaphragm by flatulence. In children the history may point to an attack of whooping-cough as the origin of the dyspnœa. In advanced cases orthopnœa is present and the patient may not be able to lie down. Cyanosis may be more marked than the degree of dyspnœa would lead us to expect. Hæmoptysis may occur and may be severe, though usually unimportant. Cough and expectoration are related to the accompanying bronchitis and are particularly troublesome in cold, wet weather. The appetite is poor, digestion bad and the general nutrition much below par.

Senile emphysema is associated with a history of general senile atrophy. The ability for exercise is limited and the respiratory necessities consequently less. Dyspnœa is therefore not marked. Paroxysmal dyspnœa is uncommon, though the associated bronchial inflammations may be attended with shortness of breath. Local and acute emphysema have only such history as is associated with the conditions which cause them. With a history of severe disease of one lung or pleura with loss of functional ability we may have a general emphysema of the opposite lung which is essentially compensatory in nature and yet may ultimately differ in no way from the ordinary morbid developments in the large lunged variety of emphysema.

Sudden and urgent dyspnœa following tracheotomy, with or without symptoms of pneumothorax, or severe dyspnœa after violent attacks of coughing or in advanced stages of general emphysema, may indicate the development of the interlobular form of emphysema.

SYMPTOMS AND DIAGNOSIS.—The symptoms of emphysema are generally modified by those of the associated lesions of the chest. The dyspnœa, cough, cyanosis, general appearance of the patient and the conformation of the chest are characteristic in the well developed forms of the disease.

The general symptomatology of the so-called hypertrophic form of the disease is well illustrated in the following history of an out-door clinic case:

M. D., wood turner, aged 49. Always healthy until five years ago, when he had a severe attack of bronchitis during the winter. The following winter had another attack of bronchitis and since then has suffered from more or less dyspnœa and cough with frothy expectoration. He is usually worse during the winter months. Has never had any bloody expectoration. At present suffers from dyspnœa which, at times, is paroxysmal and distressing and is always worse on exertion. Cough is more or less troublesome and is attended with scanty,

The barrel-shaped chest of emphysema. (After Anders.)

viscid expectoration. Has no pain, but there is a sense of constriction about the lower portion of the chest. The pulse is small, weak and at times irregular. Temperature normal, appetite poor, digestion bad, sleeps fairly well.

The patient is slightly cyanosed. The cervical veins are distended, but there is no jugular pulsation beyond that conveyed from the carotid artery. The facial lines are distinct and his face has a careworn expression. The alæ nasi are

widened, the lips thick, the eyes prominent and the conjunctivæ injected. The finger ends are clubbed. The abdomen is prominent, the liver and spleen lower than normal and somewhat enlarged from congestion. There is no œdema or ascites.

His chest is enlarged in all its diameters, particularly in the antero-posterior. It is rounded (barrel-shaped) in form with the shoulders elevated and the scapulæ rotated somewhat on their axes, the posterior borders and lower angles standing well out from the chest wall. The whole chest looks shorter though the vertical diameter is increased by downward displacement of the diaphragm, and the oblique diameter by the more horizontal position of the ribs and the widening of their interspaces. The clavicles are well forward and the inspiratory muscles of the neck are prominent and tense. The junction of the maubrium with the body of the sternum (angulus Ludovici) is prominent and the lower portion of the sternum slightly depressed. The costal angles are widened. The suprascapular depressions are effaced, the upper intercostal spaces are even with the ribs, but the lower spaces are depressed and there is inspiratory recession of these spaces below the fourth interspace; when he coughs the spaces bulge slightly. The respiratory excursion of the chest is limited and the expiration is much prolonged. The heart impulse is absent from the usual situation and there is systolic epigastric impulse. A few dilated veins are present along the anterior and lateral portions of the chest corresponding to the attachments of the diaphragm.

On palpation we find the fremitus barely perceptible over the upper chest. Below the level of the fifth rib in the axillary line the fremitus is increased on both sides. The cardiac impulse is not felt in its usual situation. The percussion note is hyper-resonant, low in pitch, empty in quality and of great intensity, even over the areas of increased fremitus and is not appreciably modified by inspiration or expiration. The resonance extends in the mammillary line to the eighth rib on the left and to the sixth rib on the right. Behind it extends to the eleventh rib. The respiratory murmur is feeble and suppressed over the upper portion of the chest, below the fourth rib it is somewhat harsh. The expiration is about three times

as long as the inspiration and over the areas of increased fremitus it is slightly higher pitched. Laterally and in front, crackling rales are heard below the fourth ribs. Behind, at the bases, the rales are finer. The heart sounds are weak and distant; the first sound, having lost its muscular tone, is short and indistinct. The second sound, over the pulmonic area, is markedly accentuated.

In senile emphysema dyspnœa is usually not severe except during exertion; bronchial and asthmatic symptoms are not very pronounced. The subjects are emaciated. Venous congestions, cyanosis, clubbing of the fingers, displacement of the heart and diaphragm are absent. The barrel shape of the chest is not marked, though the lateral diameters are diminished. The ribs are oblique and the interspaces are wide above, while below they are very narrow. Inspiration is shallow and there is inspiratory recession of the interspaces. The percussion note is low in pitch, but clear in tone and empty in quality. The respiratory sounds are feeble and the expiration but moderately prolonged. There may be rales at the base of the lungs.

In localized emphysema the signs are modified by the conditions which cause it. These conditions generally result in more or less condensation of local areas of lung tissue largely from interstitial increase of tissue. These areas, surrounded by emphysematous tissue, will give marked increase in fremitus with a low-pitched percussion note of empty quality. This apparent anomaly in physical signs is due to the more dense tissue being surrounded with alveoli in which the air tension is greatly increased.

When signs of local emphysema are present in an apex of the lungs they may be due to compensatory emphysema developing as a result of the changes incident to a healed or quiescent local tuberculosis.

Enlargement of a portion or the whole of a lung as a result of extensive disease or contraction of the opposite lung may, according to Fowler, be regarded as a true hypertrophy,—if the functional activity be increased (puerile breathing), or as emphysematous if the activity be decreased (feeble breathing with prolonged expiration).

Acute vesicular emphysema is characterized by the signs of marked distension of the chest with air, urgent dyspnœa, perhaps cyanosis and such additional signs as may attend the cause of the attack.

The symptoms of interlobular emphysema are diminished respiratory sounds in connection with urgent dyspnœa. Signs of pneumothorax and collapse of the lung, or perhaps of general subcutaneous emphysema, may be present.

The diagnosis of emphysema is usually not difficult. In pneumothorax the affected side is enlarged, the spaces obliterated, movement is restricted or absent and is exaggerated on the opposite side. The percussion note is more amphoric or tympanitic in quality and the breath sounds more indistinct than in vesicular emphysema. It may often be difficult to estimate the exact status of cases of combined emphysema, bronchitis and failure of the right ventricle, but treatment will often aid us in this respect. Aneurisms of the transverse portion of the arch of the aorta may present bronchial and dyspnœic symptoms, but the presence of local dullness or the absence of hyper-resonance, together with the brassy cough and tracheal stridor, will aid differentiation.

TREATMENT.—Emphysema, when atrophy of the septa has occurred, is not a curative affection. The future of the case must be judged by the condition of the heart and general nutrition. Most of the patients are under treatment for some of the associated affections, for, to the emphysematous state itself, they become largely indifferent because of its gradual development and progress.

The direct treatment of the emphysematous condition by means of compressed air baths (Williams) or by the inspiration of compressed air and expiration into rarified air (Waldenburg), while productive of increased comfort and some benefit to the patients, is not directly curative and involves the application of troublesome methods of mechanical treatment. Emphysematous subjects should seek such localities as furnish atmospheric conditions and air pressure suitable to their impaired respiratory power. Many cases do well at the sea level though the changes in temperature in winter and the dampness may aggravate their condition. The tendency towards

bronchitis and asthmatic dyspnœa renders a warm, dry climate the most advantageous. The altitude must be decided by the condition of the right heart and the respiratory capacity. As failure of the right heart is the chief thing to guard against, a permanent residence at a considerable altitude is dangerous.

The general nutrition of the patient must be increased by diet and tonics. Iron, arsenic and strychnia are the best tonics. Iodide of potassium in from five to ten grain doses is very useful. Iodide of sodium may be used in place of the potassium salt. A useful combination is the following:

℞

Ferri arsen.,	gr.	$\frac{1}{8}$.
Ext. nucis vom.,	gr.	$\frac{1}{2}$.
Peps. puræ,	gr.	i.
M Ext. cascara sag.,	gr.	$\frac{1}{2}$.

The various remedies already detailed for use in bronchitis and asthma are to be used when these conditions are associated with emphysema and as these are the complaints with which patients come for relief, such remedies constitute a large part of the therapeutic measures called for in emphysema. The functions of the liver and bowels must be watched. Occasional doses of mercurials are of great service at times. When congestions and œdema appear, digitalis is to be used as in other conditions attended with weakness of the heart. Strychnia in large doses is very useful in these cases.

With paroxysmal attacks of dyspnœa morphia should be used with caution if there is much bronchitis present. With marked cyanosis from an overtaxed right ventricle venesection may be indicated.

PULMONARY COLLAPSE.

Collapse of the lung may be congenital (atelectasis) or acquired. The term atelectasis was formerly reserved for cases of partial or complete failure to expand the lungs at birth, while collapse referred to the airless condition of lung tissue which had exercised function. The two terms are largely used

synonymously at present, but we shall use the unqualified term collapse as referring to the acquired variety. Undoubtedly, in some instances of the congenital form, the term atelectasis is etymologically more correct.

ÆTIOLOGY.—Congenital collapse occurs from obstruction of the bronchi by mucus, meconium or the pressure of enlarged bronchial glands. Enlargement of the abdomen may cause collapse by interfering with the action of the diaphragm, or collapse may occur from congenital disease, muscular weakness, or from mechanical injury during labor. Collapse of the lung occurs from bronchial obstruction, from bronchitis or broncho-pneumonia, especially in connection with whooping cough or measles, from diphtheria of the bronchi or trachea, fibrinous bronchitis, bronchiolitis, œdema of the lungs, thoracic aneurism, mediastinal tumors, bronchial stenosis, bronchiectasis (from mucous obstruction), pulmonary embolism, thrombosis and infarction. The various diseases and morbid growths of the nasal, pharyngeal or laryngeal passages may, if sufficiently severe or prolonged, cause collapse of the lung. Pressure on the lung from pleural effusions or on the diaphragm from abdominal distention may cause collapse. An opening into the pleural cavity is always followed by more or less collapse of the lung. Pneumothorax and pyopneumothorax are followed by collapse more or less complete. Great weakness or paralysis of the inspiratory muscles or of the diaphragm, such as occurs in severe or prolonged fevers or in weak and badly nourished, rachitic children, may cause collapse of the lower portion of the lungs. Weakness of the intercostal muscles, with partial collapse of the lower portion of the lungs during the first year of life, produces contraction of the lower portion of the chest with "pigeon breast," and other deformities of the costo-sternal articulations.

The generally accepted view as to the mechanism of the production of collapse of the lung is that it is due to absorption of air from portions of lung tissue whose bronchi are obstructed by secretion or other causes. The absorption occurs through the agency of the blood vessels, alveolar walls and the elasticity of the lung tissue (Lichtheim). In collapse occurring without bronchial obstruction, it is supposed to be due to

paralysis of the inspiratory muscles (Fagge, Pearson—Irvine).

MORBID ANATOMY.—Areas of collapse are usually more marked in the surface of the lung than in the deeper parts. The margins of the lower lobes along the vertebræ, the middle lobe of the right lung and the peripheral portions of the upper left lobe are the usual sites for collapse. The collapsed areas present as irregular, depressed, bluish-red or violet colored masses which are smooth and resistant, do not crepitate, and which sink in water. Serum may be expressed from the tissue which is usually somewhat congested. If the collapse has not existed very long the area may be dilated by insufflation, when it will be red in color and crepitant. Numerous small areas or large tracts may be affected according to the extent of the cause. In complete collapse of a lung, as from pleural effusion, the organ is heavy, airless, of a bluish-gray color and less shiny than the lobulated variety.

A collapsed portion of lung may regain its normal condition, may become œdematous and congested (splenization), or, in compression collapse, when comparatively large areas are involved, the air is gradually expelled or absorbed and the tissue becomes dry, tough and flesh-like (carnification). When the collapse is permanent the alveoli are compressed and obliterated. Interstitial tissue developments occur. The small bronchi may dilate and may contain pus. Atrophic changes may occur and a local mass of indurated tissue may remain.

Extensive congenital collapse may cause sufficient obstruction of the pumonary circulation to prevent the closure of the foramen ovale and the ductus arteriosus and cause dilatation and hypertrophy of the right heart.

CLINICAL HISTORY.—In congenital collapse there may be a history of sleepiness and inability to nurse, feebleness and arrested growth, coldness, lividity; cyanosis, asphyxia or convulsions may be present if some congenital lesion of the circulation complicates the case. The history of collapse of the lung is the history of its ætiological factors.

SYMPTOMS AND DIAGNOSIS.—In congenital collapse the child is feeble, cries weakly, the temperature is sub-normal, the breathing slow and feeble. There is inspiratory recession of the interspaces and the lateral regions of the chest.

The lower chest is contracted and the sternum prominent. The breath sounds are feeble all over the chest and, perhaps, absent at the base except when the child cries or coughs. Fine rales may be heard at the base. If much cyanosis is present the child will probably not live many days.

Collapse of the lung may be attended by sudden and severe dyspnœa, rapid and feeble pulse, cyanosis and unconsciousness, or, if the area be small, no particular symptoms may be present. Inspiratory retraction of a portion of the chest wall, dullness on percussion or a hard, tympanitic note if emphysematous patches have developed; feeble respiratory sounds; tubular or bronchial breathing; fine rales heard only after a deep inspiration where breath sounds are absent (partial collapse), are the principal signs of collapse of the lung. These will be associated with the various physical evidences of the pleural and pulmonary causes of collapse.

Collapse from pleural effusion may be mistaken for pneumonia. There may be tubular breathing, but it is more distant, the vocal sounds and fremitus are less marked or absent as are the fever and sputa of pneumonia. The percussion note is more flat, and position may alter the level of the flatness. In miliary tuberculosis the previous history of cough, fever, loss of flesh, and the family history, will aid in differentiating.

TREATMENT.—In atelectasis in the new-born the fauces must be cleared of mucus. Breathing may be established by placing the child in a hot bath and splashing it with cold water, or switching it with a wet, cold towel. Artificial respiration, insufflation of the air passages, irritation of the fauces, stimulating liniments, etc., may be used. The child should be kept warm under all circumstances. If the incubators now in use in maternity hospitals are not to be had, the child should be well wrapped in cotton-wool when not in the warm bath.

Pulmonary collapse from bronchial obstruction and pulmonary inflammation is to be treated with the remedies applicable to such causes. It is sufficient to mention the possible danger of opium in bronchitis or bronchiolitis in adding to the possibility of collapse from mucus obstruction, and also to the advantage of emetics, in the same conditions, in preventing collapse by the removal of secretion, though they must be given with judgment.

In collapse from paralysis of the muscles of inspiration and of the diaphragm, artificial respiration may be of considerable benefit. In collapse secondary to indurations of lung tissue, thickening of the visceral pleura, pleural adhesions and effusions; pulmonary gymnastics, deep breathing, compressed air inhalations, residence at a moderately high altitude, are some of the means of promoting lung expansion and developing compensation, if not actual restoration of function in the collapsed lung.

CHAPTER X.

PULMONARY CONGESTION.

The bronchial circulation is subject to the same modifications as occur in other portions of the general circulation. The mechanism of circulatory changes in the pulmonary vessels has, however, caused much discussion. It is generally conceded that the pulmonary vessels are influenced by the vaso-motor nervous system and that congestion of these vessels may result from vaso-motor disturbances. Obstruction to the passage of blood through the left side of the heart, obstruction to the general circulation, and degeneration of the blood may induce congestion of the pulmonary circulation.

Congestion of the lungs may be *acute* (active) or chronic (passive). The so-called *compensatory hyperæmia* is a form of active congestion. Varieties of chronic congestion are *brown induration* and *hypostatic congestion* or *splenization.*

ÆTIOLOGY.—Active congestion occurs in the first stage of pneumonia and in all inflammatory affections of the lungs; in rapid heart action from violent exertion or excitement; from vaso-motor paresis; from drinking large quantities of alcohol; from sudden change in air pressure in croup, laryngitis or whooping-cough; from the rarified air of high altitudes, or from the sudden removal of large pleural effusions. Active congestion also occurs in conection with infarctions of the lungs, capillary embolism of the pulmonary arteries, acute dilatation of the heart, acute myocarditis in connection with acute diseases and in rapidly developed cardiac failure in chronic cardiopathies.

Chronic congestion results most frequently from the obstruction incident to chronic heart lesions, particularly mitral disease. It occurs in typhus fever, typhoid fever or measles (splenization), or in any disease marked with severe blood changes. In bed-ridden subjects, from any chronic disease,

congestion may develop in the lowest portion of the lungs (hypostatic congestion). In mitral disease the congestion is especially liable to assume the characteristics of brown induration.

MORBID ANATOMY.—In *acute* congestion the pulmonary capillaries are prominent, the alveolar epithelium becomes swollen and granular and may proliferate. Red blood corpuscles may be found free in the alveoli. The lung tissue is red and contains an excess of blood. Red, frothy fluid flows from the cut section. Œdema may be present. According to Fowler the color of the tissue depends on the degree of aëration of the blood at the time of death.

In *chronic* congestion the lungs are heavier than normal, are of a dark-red, bluish or black color. The congestion begins in the lower lobes first. Dark blood flows from the cut surface of the lung. Œdema and extravasation may occur in the interstitial tissue. The alveoli contain swollen and granular cells and fibrin and leucocytes may be found (Cornil and Ranvier). In hypostatic congestion or splenization there is interstitial œdema and scattered red or yellow foci due to blood extravasation or to broncho-pneumonia. The tissue is homogeneous, firmer, contains little air and there is alveolar collapse. When the tissue is airless and tough it looks like muscle tissue and is sometimes called *"carnified lung."* At times the tissue is friable, there is collapse, œdema and mechanical hyperæmia, constituting a condition called *"hypostatic pneumonia."*

In *brown induration* the lungs may be smaller in size if emphysema is not present. They are firmer and do not collapse. Pleural thickening, adhesions and pigmentation may be present. The lung tissue is irregularly pigmented, dry, and contains less air than normal. Brownish, serous fluid may present from the cut surface (Virchow's *"brown œdema"*). There is elongation and dilatation of the pulmonary capillaries. The epithelial cells lining the alveoli are swollen, filled with dark brown pigment and are attached to the walls or are free in the alveolar cavity. Red corpuscles may be present in the intra-alveolar exudation. The lymph spaces of the alveolar walls contain pigment, which is also present in the interlobular peribronchial and perivascular tissues which are thickened.

There is congestion and oedema of the bronchial lining with
dilatation of its vessels. Hæmorrhagic infarction may be pres-
ent in the lung tissue. The lung is brownish, red or mottled
with brown or black specks or streaks and is more dense and
thicker than normal lung tissue.

CLINICAL HISTORY.—The history of pulmonary conges-
tion is covered by that of its ætiological factors. Dyspnœa,
cough and blood-stained, watery expectoration may be prom-
inent. In *acute* congestion there is sudden, urgent dyspnœa
with a sense of constriction about the chest, but no pain. In
chronic congestion more or less dyspnœa will be present, but
is not complained of as the patient becomes somewhat accus-
tomed to it. The temperature may be elevated.

SYMPTOMS AND DIAGNOSIS.—Increased frequency of
respiration with dyspnœa and blood-stained expectoration com-
bined with harsh respiratory sounds and fine crackling rales,
especially in the lower portion of the lungs, are the chief symp-
toms of acute congestion. *Chronic* congestion may show feeble
respiratory sounds, harsh respiration with prolonged expira-
tion and fine bubbling or crackling rales from œdema.

The ætiological associations will indicate the nature of
the congestion. Pulmonary embolism and infarction are char-
acterized by rapid, panting breathing and the expiration is not
prolonged. After the urgent symptoms of the onset of acute
congestion have passed off the breathing is not panting. In
asthma, the prolonged expiration and the characteristic rales
will differentiate. Pneumonia is excluded by the absence of
the chill, temperature, characteristic expectoration and by the
presence of its more definite physical signs.

TREATMENT.—In *acute* congestion developing suddenly
with urgent symptoms, such as cyanosis and lividity, relief
must be obtained as the right heart will fail. Dry cupping
over the entire chest or venesection (eight or ten ounces) is
demanded. Steam inhalations, turpentine stupes to the chest,
aromatic spirits of ammonia, ether, digitalis, and heat to the
extremities may be used. Large doses of digitalis are useful
in active congestion from alcohol, and alcoholic stimulation
may be advisable in acute congestion from sudden cardiac fail-
ure in acute fevers and myocardial disease. The bowels should
be kept freely open with saline purgatives.

In *chronic* congestion the treatment will vary with the nature of its cause and need not be repeated here.

PULMONARY ŒDEMA.

Pulmonary œdema is a secondary affection which is usually combined with more or less congestion of the lung of which it may be the result. An "acute" form of pulmonary œdema occurs in which its secondary nature may not be determined. Acute œdema with non-febrile onset and rusty, watery expectoration was styled pneumonia serosa by Traube.

ÆTIOLOGY.—Œdema of the lung occurs in connection with pneumonia, bronchitis, emphysema, nephritis, the various diseases of the heart, scorbutus, purpura, anæmia and heart weakness in infectious fevers. Obstruction in one part of the pulmonary circulation may cause œdema in another portion. In cases of general dropsy pleural adhesions on one side of the chest may cause œdema of the corresponding lung while there may be hydrothorax of the opposite side. Encysted pleurisies may cause local œdema in the adjacent lung tissue.

MORBID ANATOMY.—Pulmonary œdema is characterized by serous exudation in the alveoli and interstitial lung tissue. Pale, frothy fluid flows from the cut surface of the lung or is easily expressed; it is blood-stained if congestion be present. The lung tissue does not collapse, is pale or tinged with red, has a gelatinous appearance, pits on pressure, is heavy and may be friable.

Collapse of the lung, pneumonic conditions and various degrees of congestion may be present. Œdema occurs most often in the lowest portions of the lungs. The pleural surfaces are moist and the cavities of the pleuræ may contain some fluid.

CLINICAL HISTORY.—Rapid breathing, dyspnœa, more or less cough with watery, frothy expectoration are the principal features of the history. The dyspnœa may be sudden and severe. The temperature will be normal unless elevated from some associated cause.

SYMPTOMS AND DIAGNOSIS.—The pulse is increased in frequency, is feeble, and irregularity may mark the severe

cases. Cyanosis, lividity, distention of the cervical vessels and extreme dyspnœa marks failure of the right heart. When œdema is associated with acute congestion the lungs may be over-distended.

In most cases inspection and palpation are negative. The percussion note will be partially dull over the lower portions of the lungs. In chronic heart disease, œdema is apt to be most marked in the left side at the lowest portion of the lung. The breathing is harsh, feeble or suppressed in the lowest portions. Towards the end of inspiration, and possibly with the commencement of expiration, fine crackling rales are heard resembling somewhat a crepitant rale. Larger moist rales and occasionally dry rales are heard.

The crepitating rales of œdema are more liquid in character and not so closely bunched, as to time, as those of pneumonia. Their occurrence on both sides in the most dependent part of the lungs, together with their associations, the watery expectoration, the absence of chill, temperature, etc., will differentiate œdema from pneumonia. In bronchitis there is fever, tenacious mucous expectoration, the dullness in the lower lung is not marked, the rales are not so fine and are more widely scattered than in œdema. The persistently rapid breathing of pulmonary infarction and the localized physical signs are sufficient to distinguish it from œdema. In asthma and hydrothorax, the physical signs of these affections will be sufficient to prevent confusion.

The following case may be regarded as one of acute pulmonary œdema:

Man, aged 41 years, a baker by trade. Has a slight, aortic stenosis, with perfect compensation, which causes no disturbance. In February, '95, had a severe attack of influenza with some bronchial symptoms. During this attack the temperature was not above 101° F. On the fourth day of the influenza the temperature rose to 102.5° F. There was some pain in the chest and considerable dyspnœa. The next day the expectoration, which had been mucoid and rather heavy and tenacious, became more liquid, then watery and frothy with very slight blood stains. There was considerable dyspnœa and

cough. The greatest quantity of expectoration was, as near as could be estimated, over two quarts in twenty-four hours. The watery expectoration lasted three days and diminished gradually. Recovery ensued.

It could not be determined, in this case, that the condition of the heart had anything to do with the production of the pulmonary œdema.

TREATMENT.—The treatment of pulmonary œdema is largely that of the diseases with which it is associated. When œdema occurs with diseases of the kidneys, elimination by the kidneys, skin and bowels must be pushed. Dry cupping over the chest and lumbar regions may be used. In acute fevers the occurrence of œdema is an indication for general and cardiac stimulation. Calomel and salines are useful as purgatives. [Lemoine recommends a decoction of the inner bark of the elder (Sambucus niger) for œdema and ascites due to sub-acute nephritis. Macgregor reports good results from stro-phanthus in œdema pulmonum.] Strychnia and digitalis are the most useful stimulants in most cases. When œdema of the lungs develops in connection with diseases of the heart, more energetic use of these remedies is demanded. The occurrence of œdema at the base of the lungs during acute febrile disease generally indicates failure of the right ventricle and is an indi-cation for large doses of strychnia which should be given hypo-dermically (1-20 to 1-16 gr. every two to four hours as neces-sary). Strychnia is here a temporary stimulant and should be pushed. In chronic cardiac disease œdema is not so amenable to strychnia and we depend more on smaller doses combined with digitalis.

PULMONARY INFARCTION.

Infarction of the lung (*pulmonary apoplexy, embolic pneumonitis*—Jurgensen) is a circumscribed form of pul-monary hæmorrhage. Some writers describe under the head of diffuse pulmonary infarction, hæmorrhage into the lung substance from rupture of aneurisms, vascular erosions from cancer, abscess, gangrene, traumatism, or very large infarc-

tions.   The true hæmorrhagic infarction such as occurs from
aseptic embolism of the pulmonary artery, is the form under
consideration.

ÆTIOLOGY.—Infarction of the lung occurs most fre-
quently in cases of mitral stenosis or regurgitation where long
continued and marked increase in pressure causes rupture of
small pulmonary veins or capillaries (according to Ganurtz
these infarcts are due to the rupture of newly formed vessels).
Embolism and thrombosis of the pulmonary vessels cause in-
farction of the lungs.

Considerable difference of opinion has been expressed as
to the effect of embolism and thrombosis on the organ in which
they produce infarction.   The frequent presence of thrombi
in the right heart, the presence of an embolus in the largest
artery entering the infarction and the resemblance of these
infarctions to those of the spleen and kidneys which are known
to result from embolism are facts in favor of the embolic origin
of pulmonary infarctions; atheroma of the pulmonary artery,
thrombosis of the main artery entering the infarction (with-
out embolus) and the formation of infarction during great
slowing of the blood current are facts in favor of thrombosis.
On the other hand, all these causes may present without infarc-
tion, as infarction may occur without any of these factors.
Again, the experimental production of embolism in animals
does not result in infarction.

Embolism does not always cause infarction as the circu-
lation may be restored through the bronchial arteries or by
anastomosis in the capillaries of adjoining lobules (Virchow,
Kuttner).   It is evident from these considerations that infarc-
tion of the lung may arise from either of these conditions and
that no one is absolutely necessary to its production.   Conheim
and Virchow state that a plug does not necessarily exist in all
cases, but Conheim says that when emboli block a terminal
artery hæmorrhagic infarction always results.   The manner in
which blood enters the obstructed area has long been in dis-
pute.   It is generally conceded that there is reflux of blood
through the veins or capillaries.   Litten claims that the reflux
of blood is through the vessels of the capsule of the organ.
Hamilton maintains that embolism has nothing to do with the

production of infarction, that it is caused by rupture of the cap-
illaries and that the wedge shape of the infarction is due to
the shape of the terminal bronchus and its air vesicles to which
area the blood is confined. Rokitansky believed that embolism
always existed in hæmorrhagic pulmonary infarction.

All the causes of embolism and thrombosis, therefore, may
cause infarction. Thus we may have pulmonary infarction
from detachment of a thrombus in a systemic vein in phlebitis,
of a cardiac thrombus in asystolism of the right heart, or of a
thrombus in the main trunk of the pulmonary artery. Vegeta-
tive endocarditis of the right heart, and, possibly, of the left
heart also, may give rise to embolic infarction of the lung.
Fat embolism, air embolism, new growths and phleboliths are
possible, though rare, causes of embolic infarction. Phthisis,
vegetative and ulcerative endocarditis, scurvy, purpura hæm-
orrhagica, gangrene of the lungs, cholera, yellow fever, typhoid
fever, typhus fever and acute yellow atrophy may be accom-
panied by infarction of the lungs.

MORBID ANATOMY.—Infarctions of the lungs occur in the
lower lobes and in the outer and lower portions of the upper
lobes. They are conical in form, somewhat irregular in out-
line and in some instances globular in shape (interior of the
lung). They vary in diameter from a fraction of an inch to
that of an entire lobe. The apex of the wedge-shaped mass is
at the point of obstruction in the artery with the base outward,
projecting, possibly, above the level of the pleura. They are
dark-red in color, firm, with well defined margins and may be
multiple and confluent. They resemble tumors and lobular
pneumonia, but are distinguished by their color, shape, position
and associations and from pneumonia also, by their well defined
limits.

In infarction the bronchioles, alveoli and pulmonary capil-
laries are filled with red blood corpuscles. Catarrhal cells are
present in the alveoli and there may be traces of fibrin. The
adjacent pleura presents extravasations or fibrinous exudation.
Old areas of infarction show pigmented areas of coarse, fibrous
lung tissue. Neighboring bronchi may be dilated. Dissection
of the artery involved almost invariably shows thrombosis with,
probably, an embolus (Fowler). In small infarctions the blood
may be expectorated and the circulation re-established.

CLINICAL HISTORY.—Infarction does not occur in rapidly fatal cases of embolism, as two or three days are required for the formation of an infarction. There may be a history of over-exertion or excitement, expectoration of small blood clots, uneasiness or constriction about the chest, increase in the dyspnœa or syncope (cardiac cases), cerebral symptoms or pain if pleurisy be present. If the cause is a septic embolus there may be chills, hectic fever, diarrhœa, and prostration.

SYMPTOMS AND DIAGNOSIS.—Infarction may be preceded by irregular, intermittent heart action in cardiac cases. The temperature may be 101-102° F. or may not be above 100° F. If the infarction is sufficiently large there may be increased fremitus, localized dullness, bronchial breathing, or localized friction sound and tenderness on forcible percussion over the same area (Head). Subcrepitant and crepitant rales may be heard. Œdema and emphysema may mask the physical signs. In septic processes the sputum may be dark from mixture with blood or the contents of an abscess cavity may be expectorated. Perforations of the pleura, pneumothorax and pleurisy may occur.

The expectoration of scanty, dark masses of blood is the most characteristic symptom. Extreme dyspnœa of sudden advent, rapid breathing, great pain, agony and fear of death marks the occurrence of extensive embolism of the pulmonary artery rather than infarction. The sputa of echinococci and of cancer of the lung resembles that of infarctions but their longer duration and associated signs with microscopic examination of the sputum will differentiate.

In the following case infarction of the lung occurred in connection with acute endocarditis and pericarditis:

Girl aged ten years. Acute rheumatism followed by dry pericarditis and endocarditis with mitral localization and extensive regurgitation. Both sides of the heart were much dilated, liver enlarged, pulse rapid and irregular, considerable dyspnœa, temperature 99.5° F. During the third week of illness the dyspnœa became suddenly worse, with some pain in the left side of the chest. The temperature rose to 102° F., pulse more rapid, some cough but no expectoration.

Examination of the chest showed a spot, in the anterior axillary line about the level of the fourth rib on the left side, which presented dullness, bronchial breathing and a localized friction sound. These signs presented over an area about an inch and a half square. Subcrepitant rales were subsequently heard in this area and all local signs disappeared in about a week. The child subsequently died from dilatation of the right heart.

TREATMENT.—The treatment of infarction of the lung is largely expectant. Absolute rest is imperative. In cardiac cases stimulants and digitalis are necessary. Strychnia is the best stimulant. Codeine or small doses of morphine are useful for the dyspnœa (Gerhardt recommends musk and alcohol for the heart and morphine for the dyspnœa). In collapse, stimulation to the extremities by hot applications and dry cups over the chest may be used; with great dyspnœa and cyanosis, wet cups or venesection may be demanded. In septic infarctions, stimulants, tonics and supporting treatment are indicated. Infarction of the lungs occurs so seldom apart from other more common affections, that our therapeutic measures are usually directed toward the latter and beyond symptomatic means for the patient's relief, infarction demands little attention.

HÆMOPTYSIS.

The term hæmoptysis indicates the expectoration of pure blood and does not include the rusty sputum of pneumonia, the slightly stained expectoration of combined œdema and congestion of the lung or the slightly-streaked expectoration which may occur in acute bronchitis.

The consideration of hæmoptysis as a distinct affection has no pathological justification. However, its occurrence in cases presenting no other subjective or objective evidence of disease and the clinical necessity for regarding it, for the time being at least, as the only apparent affection, together with the established precedent, must be our excuse for using the term hæmoptysis in this connection. Bleeding from the nasal or pharyngeal passages, mouth or gums (spurious hæmoptysis)

in persons with diseases of these passages, anæmia or hæmorrhagic diathesis, is not included under this heading.

ÆTIOLOGY.—Diseases and growths in the larynx or trachea may cause hæmoptysis. Its most frequent cause is active or passive hyperæmia of the bronchial mucous membrane with or without weakness or degeneration of its vessels. Ulceration of the bronchial membrane or rupture of an aneurism into the bronchi may rarely cause hæmoptysis. It is an early symptom in tuberculosis with or without bronchial symptoms. Some authorities regard the development of tuberculosis as certain when hæmoptysis occurs without any other ascertainable cause. This is too radical a view, as, in many instances, the bleeding is but an evidence of the weakness of the capillary vessels and loss of tone which constitutes the predisposition to, but does not necessitate the development of, tuberculosis. Rindfleisch says that when hæmoptysis is followed by tuberculosis, the hæmorrhage is due to vascular tubercular infiltration.

Any inflammatory disease of the bronchi or lung may be attended with hæmoptysis. Tumors of the lung, hydatids bronchiectasis, actinomycosis, pleurisy, abscess of the liver, traumatism of the chest, lesions of the mitral valve, pulmonary embolism or thrombosis, degeneration of the pulmonary artery in emphysema or senile life, vaso-motor affections, the rupture of aneurisms of the aorta or of branches of the pulmonary artery, tubercular disease of the pulmonary artery, scurvy, purpura, hæmophilia, leucocythæmia, pernicious anæmia and the malignant type of infectious diseases may be attended with hæmoptysis.

It is generally conceded that hæmoptysis may occur vicariously with menstruation (vicarious hæmoptysis) though some authors consider it doubtful in the absence of disease of the lungs. I have seen two cases in which hæmoptysis occurred every four to six weeks in young women between fourteen and seventeen years of age in whom menstruation was not regularly established and who afterward developed into perfectly healthy women without any evidence of pulmonary disease. In these cases local conditions of blood pressure, from vaso-motor disturbances, are undoubtedly concerned in the production of the vicarious hæmorrhage.

MORBID ANATOMY.—After fatal hæmoptysis the bronchial membrane will be dark-red, swollen and ecchymotic. Thick, cylindrical blood casts may be found in the bronchi of the same lung, or opposite lung if the patient has been lying on that side. The aspiration of blood into other portions of the lung causes pinkish patches in the lobules. If the hæmoptysis occurred some time before the examination no trace of the bleeding may be found. Small nodules much resembling infarctions may result from aspiration of blood into air cells. In tubercular hæmoptysis tubercular changes may be found in the walls of the vessels. In advanced tuberculosis an aneurism may break into a cavity causing gradual leakage through the clot, or there may be immediately fatal hæmorrhage. The rupture of the vessel may be caused by tubercular ulceration of the vessel wall.

CLINICAL HISTORY.—The following histories illustrate the most frequent types of hæmoptysis. The first case is one of bronchial hæmorrhage from loss of tone in the bronchial capillary vessels:

Lawyer aged 38. Height 5 ft. 10 inches; weight 148 pounds. In 1891 had a slight hæmorrhage accompanying an attack of bronchitis of moderate severity; spat up a small quantity of blood on two occasions. There were slight streaks of blood in the sputa for two days afterward. In '93 had another slight attack of bronchitis with the expectoration of a small quantity of blood. In '96 had an attack of laryngitis with some bronchial irritation and cough. During this attack he spat up clear blood on three different occasions. Examination at this time showed nothing except slight harshness of the inspiration over the trachea and main bronchi. The temperature was 99 F. for three days but subsided without treatment. Examination of the lungs in March '98 showed nothing abnormal. There had been no recurrence of the hæmoptysis, and only once of the bronchial trouble. No tubercle bacilli were found at any time in the sputum. He has gained in weight and strength and considers himself perfectly well.

The following case is one of hæmoptysis in mitral regurgitation with temporary dilatation of the right heart:

Man aged 58, seen in consultation. Had an old mitral regurgitation with fair compensation but had overtaxed his heart causing dilatation of the right side. He was a large, fleshy man and when seen by me was lying in bed suffering from dyspnœa and was somewhat cyanotic. As he could not turn in bed he had caused newspapers to be fastened to the wall on either side of him and would turn his head either way and expectorate clear blood on these papers with force and precision. The total quantity of blood was considerable and the hæmoptysis lasted several days. The lower extremities were covered with petechiæ. He subsequently recovered and was about in apparently good health when last heard from.

The following case is one of hæmoptysis in tubercular lobular pneumonia. These cases present a distinct clinical type in which hæmoptysis is an early and at times persistent symptom. It appears conjointly with the rise in temperature and continues while the blood pressure is high. It is due to the severe collateral congestion attending the lobular inflammation which latter is generally tubercular in nature:

Young man aged 24 years, clerk by occupation. Height 5 ft. 8 inches; weight 136 pounds, healthy appearance. Previous general health good with the exception of two attacks of slight hæmoptysis in connection with slight bronchial inflammation.

In March '89 had an attack of hæmoptysis. Would spit up a teaspoonful of clear blood several times in an hour. Temperature 102.5° F., pulse 120 per minute and of high tension. Examination showed bronchitis and lobular pneumonia of the upper lobe of the right lung. The acute symptoms lasted six days during which time the bleeding continued in spite of all kinds of astringent remedies, ergot internally and ergotin hypodermically. These remedies were stopped and the blood tension lowered, through aconite and the continued loss of blood, when the hæmoptysis ceased. The lung did not clear

up after the acute stage of the disease had passed. Acute tuberculosis developed. Death in six months. No tubercle bacilli were found until after the attack of lobular pneumonia.

Other similar cases could be cited in which successive attacks of lobular pneumonia occurred with hæmoptysis, eventuating in tuberculosis and in which bacilli could not be found during or subsequent to the first attack of pneumonia.

In the following case fatal hæmoptysis ocurred in a subject of pulmonary tuberculosis with a large cavity in the upper right lung. There was probably some connection between the occurrence of the hæmorrhage and circulatory excitement incident to sexual effort:

Man aged 35 years. Tuberculosis of right lung, cavity in right upper lobe. Had lost much flesh but was able to attend to business. Sept. 15th, '87 retired to bed feeling as usual. Shortly afterward had a sudden hæmorrhage, brought up a large quantity of blood, went into collapse and was dead when I reached him fifteen minutes later.

It is probable that the hæmorrhage in this case resulted through tubercular ulceration of a vessel or the rupture of an aneurism into the cavity in the lung. There was free bronchial communication with the cavity.

SYMPTOMS AND DIAGNOSIS.—The quantity of blood expectorated varies from a mere streak or clot to one or two pints. It is alkaline in reaction, bright red in color or of a venous hue if the quantity be great. It is more or less frothy and may be mixed with sputum. The statement of the patients and friends in regard to the quantity of blood expectorated should be received with caution as a half teacupful, to their eyes, usually means about two teaspoonfuls by actual observation.

When the blood has remained in the lung it is black or brown and may form moulds of the tubes and such clots or pieces may be expectorated for several days after a hæmorrhage. The hæmorrhage is usually sudden, unannounced and most often not connected with violent exertion. The subject

is made aware of it by the presence in the throat of a salty substance with or without cough. With severe cases there is pallor, faintness and collapse. The pulse is rapid, the temperature normal or sub-normal, except in pneumonic cases when it will be elevated. In all cases the temperature may be elevated after the bleeding has ceased. The mental effect on the patient produces great restlessness and anxiety.

The physical signs are usually negative. The patient should not be disturbed for examination, and percussion should not be practiced as it affords no information and disturbs the patient. Auscultation may show localized signs of liquid in the bronchial tract or harsh breathing confined to a certain area, but most often is entirely negative.

In the diagnosis spurious forms of hæmoptysis should be excluded. Examination of the larynx should be made to exclude affections of that organ. Hæmatemesis is accompanied by vomiting and gastric symptoms, the blood is dark, grumous and mixed with food. (The blood in hæmoptysis may be swallowed and give rise to hæmatemesis and melæna.) In many cases it is difficult to tell just what hæmoptysis may indicate and it is well to give a cautious opinion even though local signs of tuberculosis are not clear and bacilli are absent from the sputum, as, in a large number of cases, when blood is freely expectorated, tuberculosis has already commenced. According to Sir Andrew Clark, recurrent hæmoptysis may occur in asthmatic subjects without any sign of serious disease of the lung developing either at the time or subsequently. Newman reports cases of hæmoptysis without pulmonary lesions being found post mortem.

TREATMENT.—In the milder forms of hæmoptysis the treatment is confined to general methods in regard to the patient's common condition, with such special medication as the condition of the bronchial tract may demand. In the more severe forms such as occur with lobular pneumonia or in the early periods of tubercular infection, absolute rest in bed is the first requisite. The nervous excitement of the patient must be calmed as much as possible by assuring him of the absence of immediate danger. If cough is troublesome it should be quieted by sucking small pieces of ice or by small doses of morphine.

Hot drinks and extensive cold applications should not be allowed. If the hæmorrhage is profuse moderate doses of morphine should be given hypodermically ($\frac{1}{8}-\frac{1}{4}$ gr.). If the blood is not fully expectorated, but tends to accumulate in the lungs, morphine should not be given. The bowels should be freely moved with salines as this tends to reduce the blood pressure. Nitro-glycerine should not be given because of its stimulant action on the heart.

The use of the time-honored astringents is of little or no value in these cases, but, like salt, of which the patient's friends will probably insist on his partaking in spoonful doses, if they serve to reassure the patient and modify the pernicious activity of his friends, there can be no harm in their administration. Ergot is probably more harmful than beneficial in these cases. The blood tension is usually high, and if so the bleeding will probably continue until it is lowered through the effect of drugs or the loss of blood. Ergot maintains the vascular tension and its beneficial action, through contraction of the particular vessels involved, is decidedly problematical. The use of aconite in sufficient doses to lower the blood pressure is much more rational and effective. Two-drop doses of tincture of aconite root, with ten grains of sodium bromide, may be given every hour or two until the blood pressure is relieved and the patient quieted. The action of aconite should be carefully watched.

In hæmoptysis accompanying cardiac disease strychnia and digitalis should be given. The necessity of relieving the stagnation of blood in the lungs is much more urgent than is the danger of a moderate increase in the blood pressure, in fact the former effect can only be produced through the latter means. In some cases when the right heart is over-filled, venesection may be indicated, indeed, in the severe form of hæmorrhage accompanying lobular pneumonia with high blood pressure, venesection is the quickest way to relieve the tension and stop the bleeding. Dry cupping may be of service in some cases.

The diet should be light and easily assimilated. Milk, meat-essences, etc., are best. The after treatment involves hygienic and climatic measures as well as general tonic medication. Alcohol and tobacco should be prohibited.

CHAPTER XI.

PNEUMONIA.

Pneumonia (lobar, croupous, fibrinous) is an acute inflammation of the lungs of infectious origin. It has special ætiological, pathological and clinical features which distinguish it from other acute inflammations of the lungs. These characteristics are so distinctive that it is advisable to restrict the unqualified term pneumonia to the affection under consideration.

Pneumonia may be primary or secondary. In its primary form it is most common in the temperate zone and is usually sporadic or endemic. It may occur in an epidemic form (Sturges and Coupland), especially when accompanying or following epidemics of influenza. Pneumonia is more common in winter and spring, and the monthly variations of its incidence follow those of scarlet fever and diphtheria (Folsom). Men are more often affected than women ($2\frac{1}{2}$ to 1). While no period of life is exempt, pneumonia is less frequent in infancy than in adult life, being most common from the second to the fifth decades of life. The time-honored idea that pneumonia was a disease of robust constitutions only, was due to the fact that this type of individuals exhibit mainly the sthenic form of the disease and not the atypical forms from mixed infections or secondary inflammations.

Apparently, the death rate from pneumonia has increased since 1852, though to some extent this is due to the better classification of atypical cases, especially those which simulate typhoid fever.

ÆTIOLOGY.—Pneumonia is due to infection of the lung with micro-organisms. To regard the local conditions simply as the characteristic expression of a general disease is no more rational than to attempt to explain the symptoms and clinical course of the disease by the altered mechanical relations of the

heart and lungs without reference to the toxic conditions which arise during its course. The exact relations of the subsidiary ætiological factors of pneumonia, in lessening the resistance of the individual or in producing a favorable culture field for the growth of organisms, are questions which are yet largely undetermined.

The extent to which direct infection or contagion is possible in pneumonia, is a matter of discussion. While it is evidently not actively contagious in the sense that small-pox is, still, numerous reports indicate the possibility of direct transmission of the infection, especially when the subject has been exposed to predisposing causes.

The names of Klebs, Sternberg, Friedlander, Frankel and Klein are most intimately associated with the bacteriology of pneumonia. The work of these observers, together with that of Talamon, Weichselbaum and others, has demonstrated that pneumonia is accompanied by the presence and development of micro-organisms in the inflamed lung tissue, but that neither the pneumococcus of Frankel (Micrococcus Pneumoniæ Crouposæ, Diplococcus Pneumoniæ, Micrococcus Pasteuri,— Sternberg), Friedlander's Bacillus (Bacillus Pneumoniæ) or Klein's Bacillus (Bacillus Pneumoniæ) are invariably present in pneumonia.

Frankel's pneumococcus is most frequently met with and appears to be most pathogenic of pneumonia. It may be present in the secretions of the mouth, nose and bronchial tract of healthy individuals. These various organisms, together with the streptococcus pyogenes, staphylococcus pyogenes aureus or albus and other organisms, may be present collectively or separately in pneumonia. Again, any or all of these organisms may be impossible of demonstration. Various lesions independent of pneumonia may be caused by the pneumococcus. According to Pearce the pneumococcus is invariably present in lobar pneumonia and its complications. Its presence in pure culture in the majority of cases indicates its ætiological relations. General infection, in fatal cases, is quite frequent and therefore of considerable importance both from a bacteriological and a clinical point of view.

Exposure to cold, mechanical injury or chemical injury of the lung does not directly produce pneumonia.

Climatic conditions have much to do with the prevalence of pneumonia. From November to March is the period when pneumonia is most apt to occur. The sudden changes of temperature, and the great degree of atmospheric moisture common to this period, are particularly favorable to the development of pneumonia. The low temperature and dry, penetrating winds of high altitudes may favor the occurrence of pneumonia. Duerck has shown that in persons dead from other diseases than pneumonia, the lungs may contain bacteria similar to those found in pneumonia, especially the diplococcus pneumoniæ; that injections of bacteria into the trachea of animals was not followed by pneumonia unless irritating substances were also injected, which latter, on exposure to cold alone, also caused pneumonia. Eshner thinks the harmful effect of cold depends on the resulting hyperæmia of the lungs. The fact that the pneumococcus can lose its virulence and then revert to its original type, biological characteristics and virulence (Kruse and Panisi, Eyre and Washbourn) may explain some features which seem irreconcilable with the bacterial origin of pneumonia.

Overwork, bodily injury, mental exhaustion, poor food, alcoholism, etc., may predispose to the development of pneumonia. Bright's disease, typhus fever, typhoid fever, erysipelas, rheumatism and influenza are the diseases most frequently predisposing to secondary pneumonia. During the last few years influenza has been especially prominent as a factor in the causation of pneumonias which have been marked by the atypical nature of their course and the mixed character of their infections.

Pneumonia is apt to recur in persons who have once been affected with it.

MORBID ANATOMY.—Pneumonia occurs more often in one lung than in both, the right lung being most often affected (2 to 1). In the majority of cases the disease begins in the lower portion of the lower lobes and spreads so as to involve a portion, the whole, or even more than a single lobe. It may, however, begin at any point and involve any amount of tissue. It is frequently confined exactly to the limits of a single lobe. The middle lobe of the right lung is seldom affected except in

association with other portions of the same lung. In children the apices are affected more frequently than in adults, the left lung being also more often involved.

The pleura over the inflamed area is usually involved and inflammation of the pericardium, bronchial glands, mediastinal tissue, peritoneum and meninges are frequent accompaniments.

The inflammatory process in pneumonia consists of three stages; the stage of engorgement; the stage of red hepatization; and the stage of gray hepatization.

Engorgement. In this stage the lung is exceedingly vascular and there are the ordinary changes in the vessels characteristic of inflammation. The lung is dark-red and its specific gravity and absolute weight are increased; its elasticity is lessened, it is less crepitant and more friable than normal and pits on pressure. Sticky, frothy, reddish fluid exudes on section.

Red Hepatization. In this stage there is exudation of liquor sanguinis and migration of blood cells into the lung tissue. Extravasations of blood may occur from the rupture of small vessels. The exudation coagulates in the air cells and terminal bronchioles forming a semi-transparent coagulum entangling leucocytes and red corpuscles in its meshes. The fibrin filaments are most numerous in cases due to the diplococcus pneumoniæ (Weichselbaum). Mononucleated leucocytes are as numerous as multinucleated, and pneumococci may be present in both.

The lung is increased in size and may show the impression of the ribs. It is heavier than in the first stage, solid, sinks in water and cannot be inflated. The pleura covering the inflamed area is opaque and covered with lymph. The prominent, firm, dark area of inflamed lung can be recognized before section is made. The lung tissue does not crepitate, is friable and tears with a granular fracture. The coagulations project from the alveoli and bronchi and can be lifted or pulled out. There is irregular hyperæmia of the adjacent tissue but no lobular involvement. Beyond swelling and granulation, the alveolar epithelium sustains little change as, also, do the walls aside from containing a few leucocytes. The color of the lung is dark reddish-brown, grayish or marbled.

Gray Hepatization. In this stage the leucocytes fill the alveoli and infiltrate the alveolar walls whose epithelial cells have become more swollen and granular. The tissue has a uniform appearance, having lost the granulations of the first stage. Disintegration of the fibrin, decolorization of the red cells and fatty degeneration of the white cells occur, with, in advanced cases, partial destruction of the alveolar walls.

The weight, density and friability of the lung is increased; its tissue is soft and pulpy. A puriform liquid exudes from the cut surface and the color becomes irregularly gray or yellowish white owing to fatty degeneration of the cells and to post mortem expression of blood (Rindfleisch). Advanced stages of gray hepatization have been termed "purulent infiltration" and "suppuration" of the lung.

These stages of the morbid process in pneumonia progress irregularly in different portions of the lung and may advance rapidly or slowly. The bronchi of the affected area are more or less inflamed and contain a blood-stained mucus or a dark, watery exudation if the lung is œdematous.

The blood of a pneumonic patient, when drawn, coagulates slowly. The red corpuscles having settled, the clot has a "buffy coat." This condition (hyperinosis) is not characteristic of pneumonia. A greater or less degree of leucocytosis (Piorry) is usually present. According to Cabot, leucocytosis is present early and continues throughout the febrile period; it diminishes just before or at the crisis and disappears gradually after the fall of temperature; if infection is slight and reaction vigorous, leucocytosis is slight; if infection is marked and reaction also, leucocytosis is considerable; if infection is marked and reaction is slight, leucocytosis is absent. In other words, leucocytosis may be regarded as evidence that the system is putting forth an effective struggle against the infection.

Pneumococci may be found in the blood. Their presence there gives, according to Kohn, an unfavorable prognosis as there may be a "pneumococcus sepsis" as a cause of death.

The morbid process in the lung in pneumonia may terminate in resolution, abscess, gangrene, or cirrhosis of the lung.

Resolution is effected through degeneration and absorp-

tion of the inflammatory products. It is doubtful if recovery ever ensues from advanced stages of gray hepatization. According to Weismayer the presence of streptococci should lead us to expect delayed resolution.

Abscess is a rare result of pneumonia. Pye-Smith considers it extremely rare, and undoubtedly it is, as a direct result of fibrinous pneumonia, but as a secondary result either of circumscribed gangrene or of delayed resolution from associated pleurisy or empyema, particularly in connection with mixed infections, abscess is occasionally found as a result of pneumonia.

Gangrene is very rare and occurs in debilitated subjects from coagulation of blood in the pulmonary or bronchial vessels, extensive extravasations of blood or from septic conditions of the inflammatory products.

Cirrhosis. In delayed resolution in pneumonia the alveolar walls may become involved in a new growth of fibro-nucleated tissue which may very rarely develop into a fibroid induration or interstitial pneumonia of varying extent (Addison's "marbled induration"). As a result of croupous pneumonia, however, this termination is very rare.

CLINICAL HISTORY.—While in some instances the development of pneumonia may be preceded by irregular pains, anorexia, and general malaise, and, in cases of mixed infection, the development may be slow and indefinite, in the majority of cases of typical pneumonia the onset is sudden, preceded only by headache or a nervous chilliness or shivering. Sudden pyrexia followed by sudden and severe rigor in adults, or vomiting or convulsions in children, marks the incidence of the disease. When the chill has passed the patient is prostrated and the headache may continue. The skin is hot and dry, the pulse quick, the temperature reaches from 103° to 104° F. within a few hours. Pain in the affected side may be very acute and may be the chief source of distress. It is increased by breathing, coughing or movement. There is short, irritating, hacking cough, usually most troublesome in apical pneumonias and in children. The breathing is rapid, panting in character and shallow, if there is much pain. A marked feature of the clinical history is the change in the pulse-respira-

tion rate, the normal ratio of 4-1 being altered to 3-1 or 2-1, so that a pulse rate of 90 may accompany a breath rate of 40. There is slight morning remission and evening exacerbation of temperature. The urine is scanty, dark, of high specific gravity; the urea and uric acid are increased, the chlorides diminished and it may contain albumen, blood and fibrinous casts.

The sputum is at first frothy and scanty, but soon becomes viscid and tenacious. It sticks to the mouth or lips and adheres to the sputum cup. It usually assumes a characteristic rusty-brown color, but may be thin and of a dark-brown hue (prune-juice expectoration). The sputum contains blood corpuscles, epithelial cells, mucoid cells, granular cells, oil globules, and sometimes casts of the finer bronchi. Chemically, the sputum is marked, during the febrile period, by the absence of alkaline phosphates, excess of potash over soda, excess of sulphuric acid (Bamberger), 26 per cent. of fixed salts as compared with 18 per cent. in normal mucus—the greatest increase being in the sodium chloride. During resolution these characteristics disappear.—Fowler.

The face, especially the cheeks, is flushed and of a dusky-red or purplish color. In some cases dyspnœa and cyanosis may be marked, though the latter may be present without the former.

In typical cases, for a period of from five to ten days, there is little change in the patient's condition beyond cessation of pain and headache, lower tension of the pulse, less flush in the cheeks, disturbed sleep with irregular delirium, brown and dry tongue with dry and fissured lips, more or less tenacious sputum and in some cases a small collection of herpes labialis or facialis. At any time from the third to the tenth day, but usually from the fifth to the eighth day, there occurs the "crisis." The crisis occurs most frequently on the seventh day from the initial rigor (24 per cent.), on the fifth day in 15-16 per cent., sixth or eighth days in 12 per cent., ninth day in 10 per cent., and in all cases between the fifth and ninth days in 72 per cent.—Fowler.

The first sign of the crisis may be a great change in the general aspect of the patient, a transition from great anxiety,

restlessness or delirium to calmness and assurance of recovery such as occurs so suddenly in no other disease. The patient sleeps; the skin becomes moist or there may be profuse perspiration; the temperature falls to 98° or 97° and the pulse to 60 or less; dyspnœa and delirium disappear. The temperature rises on the following day to 99.5° or 100° and then falls to normal or slightly below for the first days of a convalescence which usually occupies from two to three weeks.

This typical clinical course is subject to very great variations in the various clinical types of identical infections, infections of different nature, mixed infections and from complications.

Among the most dangerous of the irregular forms of pneumonia in which special forms of infection cannot be demonstrated are the secondary pneumonias which complicate typhoid fever, typhus, relapsing fever, small-pox, erysipelas, septicæmia and sometimes diphtheria or scarlatina, nephritis, diabetes, alcoholism, cardiac disease and those which occur after surgical operations. In many of these instances the pneumonia can be demonstrated as of a septic type, but often this cannot be done. These pneumonias are marked by absence of rigor, gradual and irregular rise in temperature, absence of pain though pleurisy may be observed on physical examination, absence of expectoration and of the characteristic condition of the urine. They are essentially latent in type, convalescence is protracted and the mortality rate is high.

Primary pneumonia may also show great variation in its clinical course in the character of the onset which may be marked by great restlessness or delirium, especially in debilitated or alcoholic subjects or in apical pneumonia, especially in children. The expectoration may be muco-purulent, greenish or bloody (hæmorrhagic pneumonia), especially in old people.

The crisis may occur with slight fall of temperature which rises again to its previous height and defervescence may occur by lysis or there may be no critical period.

The temperature may rise gradually or suddenly and then vary from 101° to 103°, defervescence being by lysis. Great variation is shown in the temperature charts of irregular

cases. In some degree the temperature shows the vital resist-
ance of the individual.

Gastro-intestinal symptoms may be marked. Vomiting
at the onset is common in children. Diarrhœa may be trouble-
some at the outset or at the crisis, especially in septic pneu-
monia.

Among the types of pneumonia which present character-
istic departure from the ordinary course of the disease is *latent
pneumonia* which occurs in debilitated subjects and in the aged.
The clinical course of this type and the physical signs are so
indefinite that a diagnosis is often deferred until post mortem.
Migratory pneumonia is a form of the disease in which there
is successive invasion of other portions of the lungs co-inci-
dently with resolution in the portions primarily affected, each
invasion being marked by the usual clinical features of the
disease. *Epidemic pneumonia* is met with apart from the
influenza pneumonias (Fox, Whitelegge, Sturges and Coup-
land). Particular epidemics are characterized by special
clinical features as a rule. *Bilious pneumonia* is a type which
is said to be associated with pneumonia of the right lung and
not to affect the course or prognosis of the disease (Pye-
Smith.) My own experience in this regard is directly con-
trary to this opinion regarding the prognosis of the bilious
type of pneumonia. According to Banti the icterus is due to
accidental hæmolytic action of the diplococcus and is therefore
hæmogenic.

The clinical course of the following case would agree
with the hæmogenic theory of the origin of the icterus.

Man aged 56 years. Height 5 feet, 8 inches, weight 215
pounds. Manufacturer. Had syphilis twenty years previous
to present illness. Subsequent health good. Present illness
of five days' duration, began with slight chill. The tempera-
ture reached 102° on the second day, but never exceeded that
point. No pain, little cough, no expectoration, moderate de-
lirium. Examination showed complete consolidation of lower
lobe of right lung. The bowels were loose, three to four small,
thin, dark, but not offensive passages daily. On third day of
illness developed jaundice, which gradually became quite

marked and of a brownish-yellow hue. The pulse was above
110 and feeble. Delirium and stupor continued. Pulse be-
came more rapid. Jaundice persisted. Death on seventh day.

Various other types of pneumonia are described, such as
typhoid, recurrent, pythogenic, cerebral, intermittent, ephem-
eral, alcoholic, senile, etc., but they have no features not com-
mon to irregular forms of any of the regular types of the
disease.

Among the irregular forms of pneumonia which are due
to a different form of infection and in which the pneumo-
coccus has a subsidiary part or is absent altogether, is the
influenza pneumonias and septic pneumonia. Since the ad-
vent of influenza in epidemic form, many cases of pneumonia
have followed an irregular course marked principally by erratic
clinical course and indefinite physical signs. The following
case may be classed as one of influenza pneumonia:

Young woman aged 24 years. Previous health good.
March 29th, 1896, was taken ill with fever and slight cough.
No chill. Temperature 101°. No pain; tongue slightly
coated, no flush on cheeks, no physical signs. On the 22nd,
slight dullness over lower lobe of left lung. On the 24th the
dullness was marked and faint bronchial breathing could be
heard. The limits of the inflamed area could not be deter-
mined. The sputum was scanty, mixed with air and never
rusty. It contained numerous organisms which corresponded
morphologically to the characteristics of the influenza bacillus.
No pneumococci were found.

The course of the disease was protracted. The tempera-
ture remained about 101° until the 3rd of April, when it began
to decline, but did not reach normal until the 10th of April.
The physical signs began to clear as the temperature declined,
but the breathing was diminished for some time after the
temperature had subsided.

Septic pneumonia may occur in connection with puerperal
or surgical sepsis or pyæmic conditions from any cause. In
some cases its origin is difficult to determine. The following
case was probably due to infection through the intestinal tract:

Professional man, aged 45 years. Height 6 feet, 1 inch; weight 210 pounds. Athletic, vigorous constitution. Had been overworking for two years. Was taken sick on Tuesday. I saw him first on Sunday. The temperature had ranged from 101° to 103°. Pulse 110 to 120. Moderate diarrhœa, slight delirium. There was no pain in the chest, no cough or expectoration. Headache was constant.

At the base of the right lung there was slight dullness and harsh breathing. The abdomen was slightly tympanitic and tender. Widal's test and the Diazo reaction were negative. By Tuesday the temperature was 104°. pulse 120, and the signs of consolidation in the right lower lobe were conclusive. There was moderate leucocytosis. The sputum was scanty, frothy and contained a very few pneumococci, a number of staphylococci and a few influenza bacilli (?). There was no special change in the patient's condition for six days, the temperature varying from 101° to 104° and the pulse from 110 to 120, the delirium continuing as did the slight cough, expectoration and irregular diarrhœa. At the end of six days the temperature began to decline and after switching between 99° and 102° for five or six days reached the normal and improvement was then slow but steady. The illness was followed by marked mental and physical exhaustion, from which recovery was gradual.

The mixed infections whose organisms are associated with the pneumococcus either from the beginning of the attack or during the course of the disease, caused marked irrregularity in the clinical history of pneumonia, as is exhibited in the following case:

Miss S, aged 22 years. School-teacher. Taken sick November the 15th with a typical attack of pneumonia. The temperature ranged, for the first six days, from 102.5° in the morning to 103.4° in the evening. The patient was very restless. On the seventh day the temperature was 101.8° and 102°, and the patient seemed better, though there was no perspiration. The pulse was five or six beats faster and the respiration about the same (45). Examination at this time

showed commencing resolution in the left lung, which had been involved. The right lung was clear. Eighth day, temperature 103°; ninth day, 103.6° and 104.2°. Examination showed that resolution was progressing in the left lung and that there was no evidence of pleural involvement. Underneath the spinal border of the right scapula there was a patch of lobular pneumonia which was not there on the seventh day. Examination of the sputum at this time showed pneumococci in abundance, a great many staphylococci and a few streptococci. On the 13th day the patient commenced to perspire, the temperature was 101.2° and 100.6°, and on the 14th day 98.8° and 97.8°. The respiration and pulse were unusually rapid at this period, being 46 and 100 respectively. A slow but steady convalescence ensued.

The complications which are most likely to modify the clinical course of pneumonia are pleural effusion, empyema, pericarditis, endocarditis, myocarditis, meningitis, parotitis, bronchitis, emphysema, valvular lesions, nephritis and, according to Bristowe, colitis.

Pleurisy is considered as part of the history of pneumonia by some observers and though some degree of pleural inflammation is usually present it is not invariably so, and for clinical purposes pleurisy may be regarded, when it can be recognized, as a complication. Pleurisy with effusion is present in about 25 per cent. of the fatal cases. Pneumococci are generally found in the exudation. The pleurisy may come on at the time of hepatization (para-pneumonic—Lemoine) and the exudation is then apt to be serous. In children and young people the pleurisy is apt to be purulent and come on at, or immediately after, the crisis. [In a recent case of apical pleuropneumonia in a child, the acute pleurisy apparently subsided, but after the crisis the temperature immediately returned to 102° and empyema developed.]

Pericarditis is a grave complication (one-half cases fatal —Huss). It may occur when any portion of either lung is affected. Pneumococci are found in the exudation. The crisis of the pneumonia is incomplete and the febrile period prolonged. Ulcerative endocarditis may complicate pneu-

monia (11 per cent. of fatal cases—Osler) and pneumococci are found in the vegetations. Acute and chronic endocarditis frequently complicate pneumonia, the latter in 17 per cent. of fatal cases and occurring most often in connection with pneumonia of the right lung. Myocarditis may be expected in connection with all severe cases of pericarditis or endocarditis associated with pneumonia, but it also occurs independently, from toxæmia, and is evidenced by œdema of the lung. The latter condition is present in fifty per cent. of the fatal cases of pneumonia. Bronchitis and empyema are frequent complications. The former may be especially dangerous.

Parotitis occurs at or after the period of crisis, usually in elderly people. Suppuration is the rule and gangrene may follow. Meningitis is a rare but usually fatal complication of pneumonia. Other complications are met with which modify the clinical history chiefly in regard to the period of convalescence.

Death from pneumonia rarely occurs before the fourth day of the disease. The period of greatest danger is from the fifth to the ninth day. When the crisis is delayed or the temperature drops a degree or two and then returns to its former height, search should be made for some disturbing element. Exclusive of infants, the death rate steadily increases as age advances. Hospital mortality is greater than that of private practice. As to children, opinions differ. Females show a greater mortality than males (in 33,606 cases; males 19.4 per cent., females 28.7 per cent.—Wells). Various statements are made regarding the general mortality of pneumonia. If statistics prove anything they show that pneumonia is a fatal disease. [The statistics of Dr. E. F. Wells— *Jour. Am. Med. Ass'n,* June 9, '92,—233,780 cases of pneumonia collected from various sources show a mortality of 18.1 per cent. Recent figures furnished me by Dr. Wells bring the total number of cases up to date to 366,544, with a mortality of 17.6 per cent.]

According to Osler the mortality statistics are uniform and, apart from complications, death is due to toxæmia or mechanical interference with the heart or respiration. In sudden death the action of the specific toxins on the heart center is more important than the effect of fever on the heart.

SYMPTOMS AND DIAGNOSIS.—A few hours after the onset of pneumonia the patient presents a somewhat characteristic appearance. He lies on his back if the disease is central or basic, or on the side if there is pleurisy and much pain. His expression, in the former instance, may be rather dull and apathetic, while in the latter case it will be anxious and he will be restless. The skin is hot and burning, the face flushed and the cheeks, over the malar prominences, will be red or purplish. The alæ nasi dilate with respiration which is short, quick and panting in character. The full, strong pulse, high temperature and short, hacking cough complete a picture of serious illness which the physical examination, at this time, fails to corroborate. In no other disease will a person be so ill and yet, apparently, have so little the matter with him, as in the first stage of pneumonia, except, perhaps, some types of influenza. Albutt says that a "sure diagnosis can be made from the aspect of the patient, the pain, the sputum, the urine and the fever, without the aid of percussion and auscultation." Nevertheless physical examination remains the only method of ascertaining the exact nature, location and extent of the disease.

Within a period, which varies with the type of the disease, of from a few hours to the second day after the onset, we can detect evidence of the morbid process in the lung. Inspection, palpation and percussion are usually negative, though there may be a slight restriction of motion, and perhaps slight dullness. On auscultation over the inflamed area the respiratory murmur is suppressed as compared with other portions of the lung. It may be slightly harsh and later becomes broncho-vesicular in character. Just before or coincidently with these indefinite signs we may be fortunate in hearing the diagnostic sign of the first stage—the crepitant rale ("le signe pathognomonique du premier degré de la péripneumonie"—Laennec). This sign lasts but a short time and is frequently not heard at all. The individuality of the crepitant rale has been denied by some observers, it being identified with pleuritic sounds by them. It is, however, entirely distinct from pleuritic friction. Crepitant rales take place immediately at the end of the audible inspiratory sound and occur as a bunch of very fine crepitations occupying but a second of time and pro-

ducing on the ear an impression of numerous, individual but
simultaneous sounds, similar to the impressions made on the
eye by the numerous but individually distinct lights of a Roman
candle at the immediate time of explosion. When heard a few
times this rale is readily recognized and is simulated only by
the rale of œdema, from which it is distinguished by, its later
occurrence and the more liquid quality and scattered occur-
rence of the rales of œdema. The physical explanation of the
crepitant rale is ever in dispute. It is generally taught that it
is due to the separation, by the air, of the walls of the terminal
bronchial passages or the alveoli. This is objected to by those
who hold that the air enters those passages only by diffusion.
They claim that the crepitant rale is a moist, bubbling rale.
It would appear that the time of occurrence of the crepitant
rale and the absence of a moist quality is not consistent with
the latter theory, for, if the air interchanges in the smaller
passages by diffusion alone, the air cannot produce a rale at
this particular time. Moreover, we would call attention to the
possibility of introducing oils directly into the alveoli by the
inspired air. (Thomas, *Jour. Am. Med. Ass'n.*—May 28,
1898.)

By the third or fourth day the signs of consolidation be-
come well marked. Motion is much decreased. Fremitus
may be increased, or absent if there is much tissue involved
and little air in the lobe. Percussion gives a dull, high-
pitched note. On auscultation the vesicular sound is entirely
lost and the breathing is bronchial in character—soft, high-
pitched and blowing in quality and distant from the ear. If,
however, a large portion of a lung is involved and the tubes
are filled with mucus, bronchial breathing may be entirely ab-
sent. Bronchial breathing is plainest during expiration, and
in central pneumonias may, perhaps, be heard only during that
portion of the respiratory act.

Bronchophony is well marked in this stage and in some
cases modified pectoriloquy may be heard. The breathing in
the uninvolved portion of the lung is exaggerated. These
physical signs, together with the high temperature, character-
istic pulse and other symptoms, continue until the crisis is
reached. When this has occurrred if we examine the lung we

find the local conditions unchanged and it appears surprising that such a remarkable change should occur in the general condition of the patient and the local condition in the lung remain apparently unchanged. The phenomenon of the crisis in relation to the local condition of the lung is an effective argument in behalf of the toxic origin of the general symptoms of pneumonia.

About, or shortly after, the crisis, examination of the lung will show moist crackling rales (rales redux) at the periphery of the portion first consolidated. More air is heard entering the affected lung, the rales spread all over the affected area and resolution is well under way. Dullness will disappear within a few days unless pleural exudation or interstitial pneumonia maintain the percussion pitch. Many modifications in the physical signs may occur. The clearness with which they present will depend on the absence of pleural exudation and adhesions and on patency of the tubes leading to the consolidated area. When the tubes are consolidated, no signs beyond dullness and loss of motion may be obtained..

The most unfavorable symptoms in pneumonia are a rapid, weak pulse, especially if the second pulmonic sound becomes gradually more short, snappy and intense; an irregular or intermittent pulse; cyanosis and rapid breathing, though cyanosis does not always depend on the mechanical obstruction in the lesser circulation, but bears some relation to toxæmia. Tympanitis, hiccough, subsultus tendinum, delirum. and hyperpyrexia are unfavorable symptoms. Apical pneumonia and double pneumonia of the bases are most likely to show unfavorable departures from the usual course of the disease.

Among those conditions whose physical signs simulate pneumonia, is pleural effusion, which is identified by the greater loss of motion, greater compensatory motion, apex displacement, loss of fremitus, flat percussion note with curved upper line of dullness, better marked sub-clavicular Skodiac resonance, absence of vocal sounds over fluid, or at least a modified form of pectoriloquy or egophony, especially in children. (Tubular breathing may be heard on the left side at the lower angle of the scapula when the lung is much com-

pressed by the fluid.) The affected side is usually increased in size in pleurisy with effusion.

Aggregated areas of tubercular consolidation may simulate pneumonia, but its course and the sputum examination will decide. Large infarctions with pleural effusion may resemble pneumonic consolidation. The erect position of the patient, the severe and sudden dyspnoea, and probably the presence of chronic heart lesion will aid the diagnosis.

The asthenic, septic form of pneumonia may be mistaken for typhoid fever. Widal's test, and the presence of leucocytosis in pneumonia, will help differentiate if the physical signs are not conclusive before these tests are reliable.

Meningitis in children simulates pneumonia before the development of physical signs in the latter disease. In meningitis the cry, the attitude, retraction of the head and abdomen, the cerebral type of vomiting, intolerance of light and the simultaneous occurrence of headache and delirium, will distinguish it from pneumonia.

Œdematous crepitation may be mistaken for pneumonic crepitation. The former is more widely disseminated, softer and more liquid in quality, and is not followed by signs of complete consolidation.

TREATMENT.—In view of the mortality rate of pneumonia its treatment becomes a most important matter. As an infectious disorder of self-limited course it is evident that the natural tendency is toward recovery which would occur in all cases if we could avoid certain contingencies. This fact is only emphasized by the low mortality rate, under widely different methods of treatment, obtained in certain series of cases by different observers. That these contingencies do not depend on the extent or stage of the pulmonary lesion is shown by the rapidity of the post-critical change in the general condition of the patient and the fact that the mortality rate shows no direct relationship to the degree of pulmonary involvement. Aside from the complications due to identical or associated infections we must consider then, that the phenomena of the disease, aside from the local condition of the lung, as well as most of its dangers, are due to toxæmia.

The phenomenon of the crisis is generally admitted to be

due to the development, during the course of the disease, of an anti-toxin, and experimental work has lately been directed towards the production of an artificial serum containing these anti-toxic properties. The Klemperers, Bonome, Emmerich and others have proved the possibility of protection from pneumococcus infection in animals by the use of immunized serum. Washbourn has succeeded in producing a standardized serum from which he reports good results. De Renzi and others report favorable results from similar serums. De Renzi had only two deaths in fourteen cases treated; Paul one death in nine; Cooke two, and Spurnell and Zanoni one successful case each. Banti and Pierachinni report no benefit in a large series of cases. While the outlook is favorable for the production of a serum with which we may mitigate the severity of the disease, if, indeed, we may not cut short its course, the matter of serum therapy in pneumonia is entirely in an experimental stage at present. The dose of the protective serum is from 10 to 20 c. c., to be repeated two or three times a day until the constitutional symptoms subside. In the meantime we must adhere to a more or less expectant plan of treatment in which routine, dogmatic methods of cold baths, hot baths, ice coils and heroic dosing of any sort must not be allowed to overshadow individual indications. Our main efforts should be to lessen the effect of the toxins on the heart and nervous system and to keep up elimination.

The diet of the pneumonic patient should be light and easily assimilated. Milk is best, but soups, broths or any of the concentrated foods may be used. The forced feeding of large quantities of food is very objectionable, as it is apt to result in digestive disturbances which interfere with the action of the diaphragm. In an adult, not over two quarts of milk should be allowed in twenty-four hours. The food should be given about every four hours.

The patient's chest should be covered with a wadding jacket cut in two sections and pinned over and under the arms.

During the first stage of the disease we must allay the general nervous and vascular excitement. Aconite and veratrum viride are the best remedies for this purpose. I prefer the former as being safer and fully as efficient as the latter.

Ten grains of sodium bromide, one-half drop of tincture of aconite root and twenty drops of liquor of the acetate of ammonia or of sweet spirits of nitrous ether, every two hours, makes a useful combination for this stage. If much pain is present there is no objection to a hypodermic of morphine or the addition of one-half a grain of codeine to the above mixture. If the skin is dry and burning and there is headache, three grains of phenacetine with two grains of sulphate of quinia (in capsules) every three or four hours will relieve these conditions as well as modify the temperature if it is high. If there is severe pleurisy pain, especially in the apical pleuropneumonias of children, a hot poultice will give much relief. There are objections, of course, to their unskillful application, but these do not apply when a little care is taken.

It is well to begin the treatment of a case with moderate calomel purgation. The bowels should not be allowed to become constipated at any stage, especially if the patient is on a milk diet, as the dry feces may become impacted in the rectum and have to be dug out. Teaspoonful doses of Husband's magnesia will be useful for a laxative during the progress of the case.

The cough and expectoration may be made easier by giving from five to seven grains of muriate of ammonia, one-sixth of a grain of codeine and twenty drops of liquor of the acetate of ammonia in syrup of tolu every two or three hours. It is well to continue this throughout the disease and to combine with it five grains of potassium acetate to promote elimination by the kidneys, the codeine being left out if necessary. Plenty of water should be allowed the patient between the hours of feeding. Tepid sponge baths should be given twice daily. The patient's mouth should be rinsed frequently with equal parts of peroxide of hydrogen and water.

The temperature need cause no alarm unless it keeps persistently in the neighborhood of 105° F., which is seldom the case if the capsules mentioned above are used. If it does we may resort to cold baths, cold applications, or ice coils as is deemed best. The heart should be carefully watched. The strain is on the right ventricle and can be best judged by the force of the second pulmonic sound. When this becomes

louder, more snappy and high-pitched, the right side of the heart needs support. This aid is best given by strychnia. It is good policy to begin the administration of strychnia early, giving 1/32 of a grain three or four times in twenty-four hours. When the right heart shows strain the strychnia should be pushed, giving 1/20 of a grain every two to four hours hypodermically. In desperate cases 1/15 of a grain may be given every two hours. One young man, seen in consultation, took, from the sixth to the ninth day, 1/15 of a grain of strychnia every two hours, night and day, and during a portion of this time 1/15 every hour for six doses. One to two ounces of whisky was also given every two hours for a portion of the time. He recovered.

Digitalis may be used conjointly with the strychnia, in which case digitalin is preferable, using about 1/20 of a grain every four to six hours. As a rule I have not employed digitalis except in cases of pneumonia associated with chronic cardiopathies. If, after the fourth or fifth day, the pulse tends to become faster and the patient restless or delirious, whisky in ounce doses should be given with the strychnia every two to four hours until the crisis is reached. I find, however, that I have used whisky very seldom. At the time of crisis if the temperature falls below 98° F., and the pulse is weak and there is profuse perspiration, a couple of ounces of whisky in hot water, dry friction of the body and a hot, dry blanket will aid matters materially.

Venesection is seldom employed at present. There are times, however, when it is useful. In the first stage of sthenic cases with a full, high-tension pulse and great restlessness, the abstraction of a few ounces of blood will give great relief. Again, in the later stage, when there is much lung tissue involved and the mechanical strain on the right heart is great, venesection may be much safer and quicker than to attempt to drain the blood into the peripheral vessels by the vaso-dilators, especially as these drugs are decidedly dangerous at this stage of the disease.

Oxygen gas is of great utility in pneumonia, both in those cases where dyspnœa and cyanosis seems to depend largely on the reduction of breathing space, and in those where

prostration is so great that the labor of breathing is telling on the vitality of the patient. While I cannot say that I have known oxygen to be the direct means of saving life, I am sure it has prolonged life in a number of instances and in others has done much towards tiding the patient over the dangerous period. Stocker recommends a more or less continuous administration of oxygen. Nitro-glycerine is a remedy which, if used at all, should be restricted to the first stage. In the advanced stages, especially if the heart is weak and toxæmia is well marked, nitro-glycerine is a dangerous remedy to use.

Among the special forms of treatment devised for pneumonia hydrotherapy has always been prominent. Cold in the form of cold baths, applications, affusions, ice pack or ice coil is variously recommended. Baruch advises tubbing in children, with the water at 95° F., reducing according to circumstances; for adults, the cold compress on front or back of chest with the water at 60° F.; if there is delirium, precede by cold affusions. Ice packs and coils are recommended by many and undoubtedly are attended with benefit in many instances. In children, however, they are more difficult to apply than are warm applications. Hayem thinks that cold baths are more powerful for good in children than in adults. Bozolo says that baths are well borne; do not produce collapse; lower the temperature for a considerable time, and give the lowest mortality rate of any form of treatment; they should be given every three hours. Hot baths have been recommended by some, but have not been much employed. A special form of the application of water has lately been used for the collapse of the later periods of pneumonia. This is the saline infusion. Undoubtedly it can accomplish good. Ewart advocates its daily use in asthenic cases, and not to wait until the toxæmia has exhausted the patient. Strict antisepsis must be observed in using the saline solution. Add boiling water to cold sterilized water until the temperature is 100° F., add one dram of salt to each pint, suspend solution sufficiently high. Use a sterile rubber tube and aspirating needle. Run the solution under the skin of the breasts, back, thigh, or abdomen. A quart can be introduced through a single tube in half an hour or so by using a little massage.

Large doses of digitalis as a routine treatment have been actively advocated by Petresco, who gives from 90 to 100 or 140 grains of the powdered drug daily. His reports are favorable both as to its action on the temperature and on the mortality of the disease. Barth advises a maximum daily dose of 45 grains of the powdered leaf in infusion. Maragliano recently claims to have established beyond doubt the specific action of digitalis on the pneumococcus. A small amount will kill the cocci in cultures and will also neutralize the toxicity of pneumonia toxins in injections. Patients are able to take larger doses of digitalis because of its effect in neutralizing toxins. If this be correct it may help to explain the seemingly inexplicable statements of Petresco.

Creosote internally (Casati) and by inhalation (Robinson), formate of soda (Rochon) and many other remedies have been specially advocated in the treatment of pneumonia, but no specific value has become attached to them.

In delayed resolution of pneumonia we may adopt the conclusions of Stengel: that in cases showing a tendency to delay, as manifested by dullness and persistent broncho-vesicular respiration systematic breathing exercises, pulmonary gymnastics, etc., are important; where marked dullness persists, counter-irritation, tonics and stimulants are indicated; the production of aseptic abscesses are of doubtful utility and often impracticable.

In the various types of pneumonia the treatment must be varied to meet the indications. We must remember, however, that the symptoms, regular or irregular, are mainly toxic in nature; that cerebral symptoms are more likely to occur in cases showing marked toxæmia, come early and have no relation to the amount of lung tissue involved.

The complications of pneumonia may modify the management of the disease as they may become, temporarily, the chief issue. They are to be managed as individual affections with due regard to their connection with the pneumonic condition.

CHAPTER XII.

LOBULAR PNEUMONIA.

Lobular pneumonia (broncho-pneumonia, catarrhal pneumonia) is an inflammation of the lung tissue occurring, as a rule, secondary to inflammation of the bronchi. Formerly it was regarded as due to direct extension of catarrhal inflammation from the tubes to the alveoli, but the presence of various septic organisms in lobular inflammation of the lungs has modified this view more in accordance with the clinical facts. Undoubtedly, in very young people, the so-called simple form of lobular pneumonia occurs from direct extension of a catarrhal inflammation of the bronchi. On the other hand we find isolated areas of lobular inflammation occurring in portions of the lungs where there has been no previous bronchitis and which can only be explained by the infection, by aspiration or otherwise, of the lobules involved.

ÆTIOLOGY.—Mixed infection is rather the rule than the exception in lobular pneumonia. The various organisms which have been identified with this form of pneumonia are the streptococcus pyogenes, the pneumococcus of Friedländer, the staphylococcus, the colon bacillus, the micrococcus lanceolatus and the tubercle bacillus. These organisms may present singly or in any combination in lobular pneumonia. [In Pearce's analysis of forty-six cases, the most frequent single infection was by the streptococcus, and the most frequently combined organisms were the streptococcus, staphylococcus pyogenes aureus and pneumococcus.]

Recognizing an anatomical and clinical distinction between bronchiolitis and lobular pneumonia, it is apparent, in view of modern bacteriological knowledge, that direct infection or irritation of the lobules, through the aspiration into them of organisms. or secretion, or both, is frequently the cause of lobular pneumonia, and that the latter occurs entirely independently of contiguous bronchial inflammation.

While lobular pneumonia is most frequent in infancy and old age, it may occur at any time of life in weak and debilitated subjects. Any influence which weakens individual resistance predisposes toward the development of this form of pneumonia. The various causes of bronchitis, irritation of the respiratory tract by the inhalation of gases, dust, particles of carbon or metals, the aspiration of food, saliva, blood or discharges of various kinds into the lungs, infection by the organism of actinomycosis, the bacillus of glanders or the specific infections of measles, whooping-cough, diphtheria or variola may cause lobular pneumonia.

Bronchitis and tuberculosis are the diseases of the lungs which most frequently cause lobular pneumonia. Tubercular lobular pneumonia is a marked feature of the clinical history of acute pulmonary tuberculosis. Pulmonary infarction, abscess, gangrene, collapse, and traumatism may cause lobular pneumonia. Pulmonary collapse, which sometimes seems to precede the inflammation, probably acts as a cause of the latter by modifying the circulation and resistance of the alveolar tissues, thus favoring infection. The exanthemata, the infectious fevers, rickets, dysentery, and extensive burns of the skin (Wilks) may be accompanied by lobular pneumonia.

MORBID ANATOMY.—Lobular pneumonia is more frequently bilateral than any other form of pneumonia. It occurs in any portion of the lungs, though, with the exception of tuberculous pneumonia, it is most often found in the lower portions. It may occur as scattered areas of lobular consolidation (disseminated pneumonia), or the aggregation of numerous areas of consolidation may resemble, clinically and anatomically, the croupous variety of pneumonia. Isolated patches involving but two or three lobules may occur.

The lesions of lobular pneumonia and its associated conditions present a great variety of anatomical changes. Bronchitis is most always present, but in some cases of tubercular or other infection by aspiration, previous bronchial inflammation of that particular area will not be found. More or less congestion, œdema and emphysema will be present. Large or small areas of collapse may be present, particularly at the base or along the free borders of the lower lobes of the lungs.

Areas of the lobular pneumonia are conical in shape, with their base outward like collapsed lobules. The base of the pneumonic patch, if at the surface, is raised, never depressed. The mass is less pliable and more nodular than that of collapse and its pleural surface is more apt to be covered with lymph. Pneumonic patches may be distinct or ill defined. They vary in size from a pea to a hazelnut. On section they are dark red or greyish with yellowish center; soft, friable, smooth or slightly granular. The patches may be discrete or form race-mose groups. When aggregated in large areas the surface of the cut lung is not as uniform as in croupous pneumonia, greater variety of stage being exhibited. Fusion of lobules, irregular peripheral distribution and slight or absent pleural exudate distinguish lobular from lobar inflammation. Fused patches of lobular inflammation may be paler, drier and firmer than usual and resemble gray hepatization.

In the early stages of lobular inflammation the alveoli contain fluid, red corpuscles and leucocytes; the alveolar epithelium is swollen and granular. The alveoli become filled with a mass of cells consisting mainly of leucocytes, in the acute form, and of epithelium in the more chronic variety. In septic cases hæmorrhagic exudation, suppuration, or sloughing and gangrene may occur. The walls of the finer bronchi are infiltrated with small round cells and their lining proliferates. Peribronchial infiltration is usually present.

Resolution through fatty metamorphosis, expectoration and absorption, is the usual termination, though this process is slower than in croupous pneumonia and may occupy a sufficient length of time to induce thickening of the alveolar and bronchial walls and dilatation of the finer bronchi. In chronic cases, fibrosis and bronchial changes may be marked. In tubercular cases caseation, encapsulation or necrosis results. Enlargement of the bronchial glands, emphysema, collapse, carnification and hypostatic pneumonia are frequent accompaniments of lobular pneumonia.

CLINICAL HISTORY.—There is no disease of the lungs which presents so varied a clinical course as does lobular pneumonia. Occurring at the extremes of life and in association with various infectious disorders; favored by toxic and septic

conditions and lowered vitality, it exhibits a complexity of physical signs and clinical symptoms closely interwoven with those of the affection of which it is a more or less intimate component part.

The onset of lobular pneumonia is not so sudden as that of croupous pneumonia, nor is its advent marked by a chill. In association with the infectious diseases of children, the advent of the pneumonia may be marked by convulsions, delirium or vomiting; more often it is announced by a more or less sudden rise in temperature, dyspnœa and cough. In old people the advent may be very insidious and marked only by great prostration and slight rise in temperature and respiratory rate; cough may be entirely absent.

The temperature range is irregular. In children it may be as high as 104°—105°. Marked remissions and exacerbations occur. In old people the temperature may not go above 101°. Defervescence is by lysis, a critical period not being observed. The breathing is rapid, shallow, and varies from forty to ninety per minute, being most rapid in children. The pulse is rapid, small and feeble, and varies from one hundred and ten to one hundred and eighty. The cough is short, hacking, dry and may be painful. Expectoration is scanty, slightly viscid and may be absent in children or old people, and in the latter there may be no cough. Dyspnœa, cyanosis and orthopnœa may be present.

Lobular pneumonia is a marked feature of the clinical history of acute pulmonary tuberculosis in which its recurring attacks mark the progress of this fatal disease. Again, the inception of the tuberculous process may be characterized by a lobular pneumonia. Hæmoptysis is a frequent symptom of these attacks of tubercular lobular pneumonia, as in the following case recently referred for opinion:

Mrs. T., aged 23. Good health until three months ago when she developed a slight cough. Three weeks ago the cough became worse, there was fever, loss of appetite and strength. Two weeks ago began to spit up blood and has continued to expectorate bloody mucus and at times clear blood. Pulse, 110; temperature, 101°. Examination showed

a well marked lobular pneumonia involving the upper third of the superior lobe of the right lung.

Lobular pneumonia may occur in connection with bronchitis and in the absence of bronchiolitis, probably through the aspiration of secretion into the lobules. The following instances illustrate this:

Man aged 38. Tinner by trade. Has a slight attack of bronchitis which has existed about a weeek. Two days before presenting himself he felt worse; his cough was drier, breathing somewhat shorter and he felt feverish. His temperature, at the time of examination was 101°; pulse, 100. Auscultation showed harsh breathing with a few mucous rales in the upper part of the right lung. In the anterior axillary line at the level of the sixth rib on the left side there was an area about the size of a silver dollar, which showed bronchial breathing with few fine crackling rales at the periphery. The respiration between this point and that of the bronchitis in the upper lobe was normal.

Examination, a week later, showed resolution to be well advanced in the small pneumonic area, while there was another area, about two inches in diameter, at the lower angle of the left scapula, which showed dullness and bronchial breathing. This area cleared up in about a week and the patient was discharged.

Boy aged 9 years. Has been troubled more or less for two years with an asthmatic form of bronchitis. Three days before presenting himself at the clinic he became worse; his cough became dry and irritating, his breathing rapid and short, there was fever and thirst, and he complained of some pain beneath the left shoulder blade. At the time of examination his temperature was 101.5°; pulse, 110. There were a few moist, and occasional sonorous rales throughout both lungs. In the lower posterior portion of the left lung there was a well defined area of consolidation about an inch and one-half in diameter, surrounded by fine sub-crepitant rales. Over this area the breathing was distinctly bronchial in character.

One week later the boy returned feeling much better. His temperature was normal, pulse, 80. Auscultation of the inflamed area showed harsh breathing with mucous rales.

The duration of lobular pneumonia is indefinite. It may last from a week to three or four weeks. If only a few aggregated lobules are involved resolution may be expected in a week or ten days. If there is succcessive invasion of segregated areas, the course of the disease may be much prolonged. This is most likely to occur in the tubercular pneumonias of acute phthisis.

Lobular pneumonia is most fatal in children before the age of five years, and when secondary to measles and whooping-cough, and in old people in connection with bronchitis.

SYMPTOMS AND DIAGNOSIS.—The symptoms of lobular pneumonia are varied and irregular. When the disease occurs in connection with measles, whooping-cough or with bronchiolitis, it is ushered in by fever, rapid pulse and breathing, possibly some dyspnœa; the cough changes, becoming dry, hacking and annoying, with scanty, more viscid expectoration.

In children, especially in connection with bronchiolitis, the rhythm of the breathing is changed; the respiratory pause being before, instead of after, expiration. There is rapid, forcible inspiration, a pause, explosive expiration immediately followed by inspiration. Short pauses in respiration may occur or the breathing may assume the Cheyne-Stokes type. When much bronchitis is present or a considerable number of lobules are involved, the breathing will be difficult and dyspnœa will be present. The alæ nasi show respiratory dilation, the accessory muscles of respiration act strongly. If bronchial obstruction is marked, the forcible action of the diaphragm causes inspiratory depression of the epigastrium. General cyanosis and lividity are unfavorable signs.

The dyspnœa does not correspond to the physical signs, as a deep-seated lesion may show marked dyspnœa and but few physical signs, while a more diffuse affair with high temperature may be accompanied by little dyspnœa (Northrup). The dyspnœa may accord with the degree of obstruction from bronchitis. When bronchiolitis is severe, dyspnœa, cyanosis

and lividity are more pronounced, the respiratory tract becomes less irritable, cough ceases and the patient seems better, but as the right heart begins to dilate there is increased cyanosis and lividity, cold extremities, inability to cough, diminished frequency of breathing, rapid, feeble pulse, dilated pupils, insensible corneæ and death by asphyxiation.

In old people the symptoms may be very indefinite. The fever may be slight, cough absent, breathing not very rapid, and dyspnœa absent if bronchitis is not severe, and only a careful physical examination will indicate the nature of the trouble.

When lobular pneumonia occurs secondary to bronchitis we may be able to trace the extension of the bronchial inflammation inwards by the appearance of small mucous, sub-crepitant and fine crepitating rales, or, the first evidence may be that of consolidation in a hitherto uninvolved area of lung. The crepitant rale is definitive of pneumonia and seldom heard in lobular pneumonia. Harsh breathing or broncho-vesicular breathing with a variety of moist rales are the usual signs of the early stages. When consolidation has occurred we may get bronchial breathing and bronchophony if the area is large enough or if the associated bronchitis does not obscure these signs. A small, superficial area of consolidation without much or any bronchitis, will give distinct signs of consolidation, while a much larger but more deeply seated area accompanied by bronchitis which obstructs the tubes will furnish very indefinite signs. Northrup warns against mistaking the tubular quality of the breathing heard between the scapulæ in dyspnœic children, for bronchial breathing. This mistake is not likely to occur if more attention is given to the pitch of the expiratory sound than to the quality of the respiratory sounds.

Percussion dullness depends on the same factors as the breath sounds, but is not so reliable, as the pitch of the note is more effectively modified by intervening areas of normal lung tissue. It is only when isolated areas of superficial inflammation, or large areas from aggregation of inflamed lobules, occur, that percussion will give reliable information. In children when numerous areas of collapse are added to lobular inflammation, and in old people when carnification of the lung, hypostatic pneumonia, lobular pneumonia and collapse

are inextricably mingled in the base of the lungs, the physical signs are decidedly indefinite, there being simply lost motion, partial dullness and suppressed respiratory sounds.

When, through the aggregation of numerous inflamed lobules, considerable areas of consolidation occur, the disease may resemble croupous pneumonia. The secondary nature, slower development, the absence of chill, lower and more irregular temperature, more rapid pulse, slower respiration, the involvement of only a portion of a lobe, its bilateral occurrence and the presence of disseminated areas of consolidation in other portions of the lung, together with the demonstration of streptococcus, staphylococcus, or a mixed infection, will differentiate.

Bronchiolitis with extensive collapse of the lung, in children, is to be distinguished from pneumonia chiefly by the development of rapid breathing, dyspnœa and cyanosis before or without a corresponding elevation of temperature. The physical signs may be practically identical. However, this condition is, as a rule, but a precursor of pneumonia and the distinction is, therefore, not important. In infants the differentiation between pleurisy and pleuro-pneumonia may be impossible except by the use of exploratory puncture. In older children a considerable degree of pleurisy is more apt to be associated with croupous than with lobular pneumonia. Enteric fever and meningitis may closely simulate lobular pneumonia.

Acute disseminated tuberculosis of the lungs may be, in the beginning, impossible of differentiation from lobular pneumonia except through the presence of the bacillus tuberculosis. A lobular pneumonia constitutes the essential anatomical lesion of this form of phthisis and the nature of the disease is determined by demonstrating the nature of the infection.

The most unfavorable symptoms of lobular pneumonia are rapid, feeble or irregular pulse, sudden fall in temperature, cessation of cough, slowing of respiration with changes in its rhythm, the appearance of stupor, delirium, cyanosis or lividity.

TREATMENT.—The management of lobular pneumonia is largely symptomatic. It is not apparent that specific treatment can be of benefit, unless, through the agency of some

serum, such as anti-streptococcus serum, we can avoid the irregular terminations which are likely to occur in inflammations depending on severe infections with pyogenic organisms.

In lobular pneumonia in children the patient should be placed in a well-ventilated room. If the cough is loose and the child perspires, the air should be fresh and dry. If the cough is tight, the expectoration viscid and scanty and the skin dry and hot, a tent may be arranged around the bed and the air be kept charged with steam by some sort of vaporizing apparatus. The diet should be milk or some of the prepared milk or beef foods. Lime-water should be added to the milk and the food should be given in small quantities every three hours. Unless pain prevents, the position of the child should be frequently changed so as to favor deeper inspiration, especially when bronchiolitis is present.

Stimulate expectorants must be given. Muriate or carbonate of ammonia, senega, etc. Two grains of chloride of ammonia, one-fourth of a drop of tincture of aconite root, fifteen drops of the liquor of the acetate of ammonium may be given every two hours to a child two years old. [Dr. Burney Yeo recommmends benzoate of soda internally, and a warm, alkaline spray containing bicarbonate of soda, glycerine and carbolic acid.] When the cough is troublesome it must be controlled with small doses of paregoric, Dover's powder or codeine. Care must be exercised in the administration of these sedatives, especially when bronchiolitis is present, as they may add to the danger of paralysis and obstruction of the bronchial tubes and consequent collapse of the lung. When cyanosis is present, especially in old people, oxygen is of great service and may be administered more continuously and effectively when warmed by passing it through a spiral metal coil placed in warm water (Fowler). Coutts advocates the use of belladonna in broncho-pneumonia in children, and is sustained by Hodghead. Small doses are slightly sedative and narcotic; they raise the arterial tension and tone of the heart. Belladonna acts as a respiratory stimulant, flushes the cutaneous circulation, increases the amount of urine and bile, checks bronchial secretion. Hodghead gives it in full doses every hour or two hours until relief is obtained. In a case reported it was given in one or two drop doses every half hour.

In children, the chest should be protected with a cotton-batting jacket. When pleurisy is present a poultice is most useful in relieving pain and nervous dyspnœa. It should be applied so as not to obstruct respiration, should be covered with dry, warm flannel and not be allowed to become cold. If expectoration is scanty and mucus obstructs the tubes, an occasional dose of one grain of powdered ipecac may relieve this condition, or, if the obstruction threatens asphyxiation, larger doses (five to fifteen grains; five grains for a child one year old) to produce vomiting, may be necessary. In asphyxiation from associated collapse of the lung, inflation of the lungs has been recommended. The abdomen is tightly wrapped with a bandage, the nose closed with the operator's fingers and his mouth applied to that of the child. This method is of doubtful utility. If the pulse and respiration become very rapid, strychnia must be given in stimulant doses and preferably by the hypodermic method. In children, from one one-hundredth to one-fiftieth of a grain may be used every two to four hours; in adults from one-thirtieth to one-twentieth of a grain at similar intervals. Many cases may be tided over the dangerous period by the energetic but careful use of strychnia.

Various hydrotherapeutic measures are recommended for the relief of high temperature and dyspnœa in lobular pneumonia. Undoubtedly these measures are the most valuable at our command for this purpose, when properly modified to suit the circumstances of the individual case. Jurgensen recommends a twenty minute bath at a temperature of 77° to 80° F., combined with cold sponging of the chest; the bath to be repeated whenever threatening symptoms reappear or the temperature reaches 103° F. Wilson Fox advises a cold bath (from one to three minutes) at a temperature of 65° to 70° F., with cold applications to the chest. Pye-Smith advises tepid sponging and tepid baths. Fowler advises, for high temperature, dyspnœa and cyanosis, hot baths (104° to 110° F.) with cold affusions (60° to 65° F.) over the chest and back. Whatever degree of cold we employ, the best evidence of its efficiency is diminished frequency of pulse and respiration. No absolute rule can be laid down based on the temperature. Children bear high temperature well, and a temperature of

103° cannot be held as abstract evidence of the necessity of a cold bath. It is rather the combination of high temperature, pulse and respiration, which indicate the employment of hydro-therapeutics. My own experience is very favorable to cool sponging frequently repeated, or bathing, for from five to fifteen minutes, at a temperature of 98° to 100° F. The child can then be wrapped in a wet towel and a blanket and allowed to rest for from fifteen minutes to an hour, when it should be gently rubbed and wrapped in a dry blanket. Northrup recommends a convenient method, of placing the nude child in a hammock made of a towel and lowering it into the bath tub, bathing its face meanwhile with the same water as is used for the general bath. This method avoids much handling of the child and is convenient. In old people, baths, if used at all, should be confined to sponging, as the exhaustion incident to the administration of baths is greater than in children.

When the circulation shows evidence of failure, digitalis and alcohol may be indicated temporarily, though these remedies will be less often necessary if strychnia is given early enough and in sufficient doses.

In the management of the convalescing period we must use general and special calisthenic exercise, deep breathing exercises, climatic treatment, nutritious diet, tonic medication, as cod-liver oil, guaiacol, iron and quinine, iodide of iron, syrup of hydriodic acid, mineral acids, in short, every medicinal or hygienic means which will increase the patient's strength and respiratory capacity and lessen the danger of secondary processes in the lungs.

PULMONARY FIBROSIS.

Fibrosis of the lung (chronic pneumonia, interstitial pneumonia, cirrhosis of the lung) is the result of a productive inflammation produced by influences of moderate severity, but of protracted course. It is characterized by gradual increase in the connective tissue of the lung and results in induration of the pulmonary tissue and obliteration of the alveoli of the lung.

It is evident that there is no disease of the lungs with which fibrosis, in some degree or extent, may not be asso-

ciated; while in some instances, as in tuberculosis, it constitutes a conservative process of nature, and a necessity to the limitation of the infective process. We are not surprised, therefore, to find the ætiological nomenclature of fibrosis of the lungs resulting in the description of several special forms of the disease which, while they may have certain more or less distinctive clinical features, have yet no generic or pathological individuality and which are not entitled to be described as special forms of pulmonary disease. Such varieties of fibrosis as pneumoconiosis, syphilitic sclerosis of the lung, etc., will therefore be considered as types of the progressive induration characteristic of fibrosis of the lungs.

The essential features of fibrosis of the lungs is the increase of the tissues of the walls of the alveoli and of the interalveolar, interlobular and sub-pleural tissues. Topographically, any one of these tissues may be chiefly affected, though as a rule they are all more or less involved.

ÆTIOLOGY.—Fibrosis of the lungs occurs secondarily to some disease of the alveolar or bronchial wall or pleura. Its most common cause is the chronic disseminated form of pulmonary tuberculosis. Lobular pneumonia, especially in early life, is frequently the cause of fibrosis starting as a thickening of the alveolar walls. Bronchiectasis, pulmonary collapse, bronchial obstruction from pressure of an aneurism, and new growths in the lung, pleura or œsophagus may induce fibrosis of the lung. In cases of protracted resolution of croupous pneumonia there may be a slight induration of the alveolar walls which in rare instances may cause extensive fibrosis of the lung.

The following case of diffuse fibrosis was probably secondary to pneumonia:

Woman aged 42. Married. Always been healthy with the exception of an attack of pneumonia nine years ago from which recovery was slow. For the last two years has had cough with gradually increasing dyspnœa on exertion. Spits up a little white, glairy mucus, particularly in the morning. Temperature 98.5°; pulse 98. Has lost some flesh. Has no pain and appetite and digestion are fair. Examination shows

marked contraction of the left side of the chest with great re-
striction of motion on that side. There is compensatory en-
largement of the right side of the chest. The heart apex is
displaced upward and to the left. The finger ends are not
clubbed. Vocal fremitus is marked all over the left side and
the percussion note is high-pitched and has a woody, hard tone.
slightly tympanitic in quality underneath the left clavicle. Very
little air enters the left lung and the respiratory murmur is
high in pitch and of a tubular quality with prolonged expiration
in the upper portion of the lung. No rales are heard except an
occasional mucous click. There is exaggerated or puerile
breathing in the right lung. No tubercle bacilli can be found in
the sputum.

In this case the length of time the disease has probably
existed; the absence of fever, night sweats, emaciation, de-
structive processes in the lung and of bacilli would seem to
refer the cause back to the previous attack of pneumonia.

A special form of pneumonia is described (subacute in-
durative pneumonia—Kidd, primary parenchymatous pneu-
monia—Buhl, Heitler) in which fibrosis of the lung is a chief
lesion.

Syphilitic gummatous growths in the lung may be asso-
ciated with interstitial fibrosis having a tendency to extend
along the vessels or tubes. There is also a diffuse form of
fibrosis occurring in children with congenital syphilis. This
form is rare and not much is known about it.

The inhalation of solid, irritating particles of dust, metals,
etc., is a frequent cause of fibrosis of the lungs. Numerous
designations have been given fibrosis from this cause. When
due to particles of carbon it has been known as *anthracosis;*
siliceous particles, as *chaliosis* or *silicosis;* of metallic particles,
as *siderosis;* of cotton particles, as *byssinosis,* etc. *Pneumo-
coniosis* is the term usually employed for this variety of fibrosis,
though such names as coal miner's phthisis, potter's phthisis,
knife grinder's phthisis, etc., are sometimes used. Any dusty
occupation may give rise to pneumoconiosis. Claisse and Josue
conclude that the lesions of pneumoconiosis are not due to direct
action of dust but to infectious processes which are superadded;

very small dust particles may migrate through the pulmonary tissues without causing disturbance; large particles produce, by traumatism, a soil upon which inhaled organisms develop; morbid conditions influence pneumoconiosis only when they are of long duration; lesions of the lymph nodes increase pneumoconiosis by impairing the circulation and the elimination of dust particles by the lymphatics; pneumogastric lesions favor pneumoconiosis by destroying the resistance of the upper air tracts and by producing vaso-motor troubles.

The intimate association of fibrosis and tubercular changes in the lungs has occasioned much doubt as to the occurrence of non-tubercular pneumoconiosis, and as the latter disease is liable to show secondary tubercular infection, it may be very difficult to determine the nature of the primary changes. It is, however, considered, by the majority of observers, that tubercular infection is not necessary to the development of pneumoconiosis.

"Corrigan's pneumonia" (interstitial) which was claimed to develop without apparent cause, Sir Andrew Clark's "fibroid phthisis," and Harris' "secondary" interstitial pneumonia are all conditions of the same inherent nature, varying, perhaps, in their ætiological factors but all manifesting the general tissue change of fibrosis of the lungs.

With empyema or in connection with long standing effusions in the pleuræ we may have fibrosis of the lungs develop either as dense bands of thickened interlobular connective tissue or as a more general fibrosis.

MORBID ANATOMY.—The morbid anatomical features of the lung in cirrhosis are necessarily varied in degree and extent. It is not necessary to review here the degrees of fibrosis that may be associated with the various pulmonary lesions.

In general and well advanced fibrosis of the lung the organ is diminished in size. The tissue is dense, firm, smooth and may be, in spots, almost cartilaginous in consistency. It may be deeply and irregularly pigmented. The alveolar structure may be entirely in places. The bronchi are irregularly dilated and may contain irritating secretions which, when not removed by expectoration may give rise to secondary inflammation, ulceration or excavation; caseous changes are absent

in no-tubercular cases. The pleura is thickened and may be adherent.

When secondary to croupous pneumonia these changes begin in the walls of the alveoli and ultimately involve the interlobular tissue. According to Green and Goodhead the intra-alveolar products of a croupous pneumonia have been known to become vascularized and develop into fibroid tissue. In lobular pneumonia and pneumoconiosis the changes begin in the alveolar walls but the peribronchial and interlobular tissues are more actively concerned in the process.

When secondary to pleurisy the tissue growth of fibrosis extends from the thickened pleura along the interlobular lymph vessels and spreads to the peribronchial tissues. The lung may be thus encapsulated or traversed by numerous bands of dense fibrous tissue.

In collapse of the lung there are hæmorrhagic and pigmentary changes, fibrosis of the alveolar walls, destruction of the epithelium and fusion of the alveolar surfaces.

CLINICAL HISTORY.—The clinical history of fibrosis of the lungs is necessarily varied. The minor degrees of fibrosis have no history apart from that of the lesions with which they may be associated and which they modify.

When fibrosis is secondary to croupous, lobular pneumonia or chronic pleurisy there is a history of protracted and incomplete recovery from these conditions. There is cough with frothy, muco-purulent or mumular expectoration. Hæmoptysis may occur even in non-tubercular cases. More or less dyspnœa will be present. Fever may not be present and will not be high unless sepsis from excavation or bronchial dilatations should occur. The course of the disease is protracted and the later stages marked by cardiac failure.

In pneumoconiosis there is a history of dusty occupation, of a bronchitis which had no definite time of onset and which is troublesome in the morning and on changing from a warm to a cool atmosphere. The expectoration is not free and consists of glairy mucus which may be pigmented. Recurrent exacerbations of the bronchial catarrh occur. Dyspnœa gradually becomes troublesome. Asthmatic symptoms may be prominent. Loss of strength and flesh occur and the patient seeks quietude in a warm atmosphere.

In syphilitic fibrosis the clinical history will differ but little from that of other forms. The cough is the earliest and most prominent symptom. Expectoration, fever, etc., have the same causal relations as in other varieties of fibrosis.

The pleurogenic form of fibrosis is illustrated in the history of the following case:

Boy aged 12 years. Good family history. When seven years old had an attack of illness, probably some form of acute pneumonia, which was followed by an empyema. The pleural fluid was aspirated several times and finally an imperfect drainage was instituted. The discharge from the pleura stopped after several months. He now suffers slightly from cough and considerably from dyspnœa on exertion. On examination we find much retraction of the left side below the fourth rib. The heart is displaced backward and upward, the stomach toward the right and downward and there is an evident degree of scoliosis. The upper part of the left lung is dilated and there is inspiratory retraction of the left side over the affected area and no air can be heard passing in or out of this portion of the lung.

SYMPTOMS AND DIAGNOSIS.—Aside from the indefinite subjective symptoms the signs of fibrosis are mainly physical. The affected side will show restricted motion in part or in whole and may be considerably smaller than the opposite side. If the fibrotic changes are restricted to the lower portion of the lung, marked contraction of this portion may be shown with compensatory enlargement of the upper portion. There may be decided displacement of the heart upwards, backwards, or to the right if the right side is affected. In extensive fibrosis marked clubbing of the finger ends may be present.

The vocal fremitus is usually increased over the affected area though it may be absent over the lower portion of the lung if the latter is very hard. The percussion note is high-pitched and of a dull wooden, or amphoric character if there are bronchial dilatations or cavities. Increased vocal resonance, broncho-vesicular breathing, bronchophony or pectoriloquy with high-pitched, prolonged expiratory sound and pos-

sibly bronchial or cavernous breathing may be heard if there is complete consolidation or cavities. Various sized mucous rales, gurgles, or mucous clicks may be heard.

The physical signs, as well as the subjective symptoms, are modified by the degree of emphysema and bronchial dilatation which may have developed.

In dust phthisis the discoloration of the sputum or the presence of foreign substances in the expectorated matter may indicate the nature of the disease.

The following case of pneumoconiosis presents the usual history:

Man 47 years old. Bohemian, married. Good family history. Wood turner by trade. Has been employed for years in a mill at very dusty work. Has coughed for several years, more in winter than in summer. Lately has been free from cough only when in a warm atmosphere. Expectoration has become more abundant, thicker and recently decidedly fetid. For the last four months has been coughing up a quantity of foul smelling mucus in the morning. He is decidedly dyspnœic and at times slightly cyanosed on exertion. His finger ends are markedly clubbed. Temperature 99.5°; pulse 100.

Examination shows retraction of the lower part of both sides of the chest, particularly of the left side. Lateral expansion is almost entirely lost. There is increased fremitus, vocal resonance and high-pitched percussion note all over both lungs. The percussion note, over the region of the fourth left rib just inside the mammillary line, is hollow in character and there are gurgles, pectoriloquy and cavernous breathing in the same region. Numerous rales and breathing of a tubular quality are heard throughout the lower two-thirds of both lungs. No bacilli in the sputum.

These cases are likely to develop tuberculosis during their course and after this has occurred a correct diagnosis may be difficult.

Besides the congenital syphilitic fibrosis of children there may occur various degrees of fibrosis and secondary conditions resulting from acquired syphilis. These conditions may or

may not be associated with gummata, or ulcerative lesions of the respiratory mucosa, and constitute the so-called cases of syphilitic phthisis.

The following case is probably one of syphilitic fibrosis of the lungs though the diagnosis rests on the absence of bacilli from the sputum, the subject being alive :

Man aged 39 years. Waiter by occupation. Family history good. Had a hard chancre eighteen years ago which was followed by sore throat and a skin eruption. Never had pleurisy or pneumonia. For the last five years has had a cough which is worse in winter and which has been especially troublesome for the last year. Expectoration which was formerly scanty and frothy has lately been muco-purulent and at times somewhat offensive. Has lost flesh and strength and there are occasional pains through the chest.

Examination shows loss of motion of the lower portion of both lungs with signs of marked induration of the left lung below the fourth rib and of the posterior and lateral portions of the right lung below the sixth rib. No bacilli have been found in his sputum. He has improved considerably, in a general way, on iodide of potassium and biniodide of mercury.

The diagnosis of localized fibrosis of the lung is not difficult and rests on the character of the physical signs. In the diffuse varieties the main question is whether they are, or are not, tubercular. This question is determined, principally, by the presence or absence of tubercle bacilli. Non-tubercular fibrosis is usually unilateral and begins in the lower lobes in contrast with the characteristic apical invasion of the tubercular form. Rare cases of primary basic tuberculosis of fibrotic nature may occur and be difficult of diagnosis but here we must remember the probability of tuberculosis occurring in connection with fibrosis which was primarily non-tubercular.

Pleural effusion which has not fully absorbed and which has prevented expansion of the lung may closely simulate fibrosis. There may be a greater degree of retraction than the same extent of fibrosis would occasion, while the voice and breath sounds will be diminished rather than increased. When

bronchiectasis is added to this condition the diagnostic diffi-
culties are increased.

Pulmonary fibrosis with bronchiectasis may be very dif-
ficult to distinguish from an empyema or hepatic abscess which
has perforated the lung. The diagnosis may rest on the result
of an exploratory puncture.

Malignant growths in the lung or mediastinum may sim-
ulate fibrosis. The latter is favored by long duration, com-
pensatory enlargement of the opposite lung, frothy, muco-
purulent or fetid (if there is bronchiectasis) expectoration and
considerable cardiac displacement. Pressure signs, dullness
extending across the median line, persistent pain in the chest
and red-colored, gelatinous expectoration are indicative of ma-
lignant growths.

TREATMENT.—In the localized fibrosis resulting from
pleurisy, lobular pneumonia, bronchiectasis, etc., deep breath-
ing, pulmonary calisthenics, general hygiene, tonics and per-
haps climatic treatment, are the means available. In diffuse
fibrosis we must employ iron, strychnia, cod-liver oil and all
the measures indicated in chronic lung disease whether the case
is tubercular or not. In very chronic cases strychnine is valu-
able as a stimulant to the right ventricle which sooner or later
will fail to keep up the lesser circulation.

Climatic treatment is very important in these cases as they
run the risk of repeated attacks of bronchitis as well as the
more serious danger of tubercular infection in non-tubercular
cases if they remain in moist, changeable climates. Warm,
dry air with sunshine, is what they need. The altitude should
not be too high,—not over twelve or fifteen hundred feet if
the fibrosis is at all extensive. The selection of a proper cli-
mate may be difficult and must rest with the patient as the
physician can only give general directions suitable to the indi-
vidual case. In a recent case of fibrosis of the lower part of
the right lung secondary to pleurisy, the patient, a physician,
had tried many localities without finding one which seemed
adapted to his case. He required warmth, dryness and a mod-
erate altitude. These conditions can only be obtained by vary-
ing the place of residence at different seasons.

In syphilitic cases the iodides and mercury should be

given. In advanced cases it may be well to omit the mercury and give iron, strychnia and cod-liver oil with the iodide of potassium. Tubercular fibrosis in a syphilitic subject should be treated mainly as a case of tuberculosis, continued mercurial courses being inadmissible. Syrup of the iodide of iron is a useful remedy in these cases.

In cases of fibrosis of the lung associated with well developed bronchiectasis accompanied by offensive expectoration the treatment should be such as has been advised for bronchial dilatation—intratracheal injections of guaiacol, creosote vapor baths, etc. Surgical interference, for the drainage of bronchiectatic cavities resulting from fibrosis, is of doubtful utility especially in syphilitic cases. In tubercular fibrosis the treatment is that of chronic tubercular disease of the lung and is considered under that subject.

CHAPTER XIII.

PULMONARY ABSCESS.

Pulmonary abscess is a more or less circumscribed collection of pus within the lung. The term abscess of the lung is frequently applied to collections of pus within the chest which have little pathological resemblance to ordinary abscess. These collections may be single or multiple. While pulmonary abscess is almost always the result of some other disease of the lung, we are justified in considering it a distinct clinical affection because of its individual features.

ÆTIOLOGY.—Single abscess of the lung occurs in connection with pneumonia, from softening and necrosis of an area of consolidated lung tissue and is favored by constitutional vices and alcoholism; also such from injuries, as fractures of the ribs or penetrating wounds of the lung. Multiple abscesses of the lungs are usually pyæmic in nature and occur from the softening of infarctions of the lung, from septic embolism of the lung and from septic broncho-pneumonia.

Pulmonary abscess may occur in tuberculosis of the lung, from the rapid breaking down of a large caseous area; in bronchiectasis with destruction of the bronchial walls and contiguous structures, or possibly without such changes; in connection with non-tubercular cavities resulting from local destruction of blood supply; from suppuration of an hydatid cyst of the lungs; from extension to the lung of suppurative inflammation of the pleura, bronchial glands, mediastinum, subdiaphragmatic space, liver, and, in rare instances, of the spleen and pancreas. Malignant disease of the œsophagus may cause abscess of the lung tissue. Circumscribed gangrene may end in abscess. The most frequent cases of abscess of the lung are those secondary to pneumonia, pyæmic infection of the lung, and to empyema.

MORBID ANATOMY.—The morbid anatomical conditions

with which abscess may be associated are very varied, but these conditions do not belong to pulmonary abscess.

Abscess following pneumonia occurs most often in the middle or upper lung. Multiple pyæmic abscesses occur in any portion of the lungs but may be confined to the base or lower portions. They are of small size, from that of a split pea to that of a chestnut, and, when at the surface of the lung, are covered by small areas of pleural inflammation. These embolic abscesses are surrounded by a zone of hyperæmia and are apt to be near the surface of the lung with their bases just beneath the visceral pleura.

In circumscribed gangrene the necrosed tissue may be expelled through a bronchus and granulation tissue may form in the walls of the cavity and generate pus. These cavities may close by granulation and cicatrization.

The expectorated matter from a pulmonary abscess consists of pus of a yellow or yellowish-green color containing elastic fibers and particles of lung tissue. There is, usually, little or no odor at first. Many kinds of organisms are present, the most frequent being pneumococci and streptococci. The discharge from an encysted empyema, suppurating bronchial gland, etc., which breaks into a bronchus, will not contain elastic fibers except at the immediate time of rupture.

CLINICAL HISTORY.—The clinical history of pulmonary abscess is necessarily varied. The principal symptoms—pain, cough, dyspnœa and fever—are common to various disorders with which abscess may be associated or of which it may be the result, and the latter may be well advanced before it is possible to individualize any of these symptoms as indicative of abscess of the lung.

Multiple embolic abscesses of the lung may give no history apart from that of a pyæmic state, with, possibly, a small amount of fetid expectoration. The occurrence of embolic abscess may be marked by sudden, sharp pain in the chest.

The history of sudden and profuse expectoration of pus, with or without pain, may be the first reliable indication of an abscess of the lung.

When abscess of the lung occurs from pneumonia the fever maintains, or returns to, a high degree and subsequently

shows a fluctuation indicative of sepsis and is accompanied by sweating and, perhaps, rigors. Dyspnœa may be troublesome but exhibits no features specially indicative of abscess, and the same may be said of the cough and pain.

The course of pulmonary abscess depends on the nature of its cause. It may be prolonged; death may occur from sepsis or from septic lobular pneumonia due to infection by material from the abscess cavity. Single abscesses following pneumonia may heal by cicatrization after the discharge of the contents of the cavity. This termination, while rare, I have known to occur in more than one instance, as in the following case:

Boy aged 18 years. Previous health good. Had a moderately severe attack of pneumonia of the right lung. After the crisis the temperature rose to 102.5° F. and showed a daily fluctuation from 99° F. to 102.5° F. with daily sweating.

Examination, six days after the crisis, showed an area in the right mammary region which exhibited dullness, suppressed respiratory sounds, increased fremitus, and rales surrounding this area. There was considerable pain in the right side of the chest. Four days later a considerable quantity of pus was expectorated which contained some elastic fibers. The patient was very weak and exhausted and continued to expectorate quantities of pus daily which became offensive. The odor was controlled by inhalations of an oil solution of menthol, aristol and eucalyptus. The temperature ranged from 99° to 101° F. The pain gradually disappeared. Pectoriloquy and amphoric voice and breathing were obtained over the area where signs of consolidation had been present.

The patient gradually improved under tonic treatment and nutritious diet, and three months later was, apparently, well; the cough and expectoration having ceased. At this time the signs of a cavity could not be obtained, there being simply increased fremitus, partial dullness and breathing of a high-pitch and of tubular quality over the area involved.

SYMPTOMS AND DIAGNOSIS.—Pain in the chest, cough, fever, dyspnœa and the expectoration of a quantity of pus con-

taining elastic fibers are the symptoms of abscess of the lung,
in most instances these symptoms are characterized by their
indefiniteness. Sudden pain in the chest followed by cough,
dyspnœa and septic temperature indicates embolic abscess,
especially in connection with septic conditions in which emboli
are liable to occur. Continued pyrexia after pneumonia, espe-
cially if of a septic type, cough and rapid respiration are sug-
gestive of abscess.

The physical signs are not always conclusive but when
taken in connection with the subjective symptoms a diagnosis
can usually be reached.

Before discharge of the contents of the cavity has oc-
curred there will be localized dullness, diminished breath
sounds, and, perhaps, increased fremitus. If there are several
areas of softening there will be crackling rales around these
areas. At this stage the absence of displacement of the apex
beat of the heart, and the presence of fremitus are important
in the exclusion of pleural effusion.

When a cavity has formed there may be a tympanitic
quality to the percussion note if the cavity is near the surface
of the lung, or the note may be high-pitched and metallic in
quality. Perhaps cracked-pot resonance may be obtained. If
the cavity has free communication with a bronchus the per-
cussion note may be higher with the patient's mouth open than
with it closed (Wintrich's change of pitch), or, if the cavity
be of greater diameter in one direction than another and con-
tain fluid, the percussion pitch will vary in the upright and re-
cumbent positions (Gerhardt's change in pitch). Again, the
rise in percussion pitch with the mouth open may be obtained
only when the patient is recumbent (Wintrich's interrupted
change in pitch).

The voice sounds may be amphoric in quality and pector-
iloquy may be obtained. Careful study of the loud and whis-
pered voice sounds is of great value. The breathing may be
cavernous in character and, perhaps, amphoric in quality.
Gurgles and metallic tinkling may be heard.

Bronchiectasis and circumscribed gangrene are the affec-
tions within the lung most likely to simulate abscess. In the
former the sputum will not contain elastic fibers, and pyrexia

may be absent, or, at least, not marked or of a septic type. The history is different from that of abscess.

In gangrene the course is more rapid, there is greater prostration and constitutional disturbance, the expectoration is fetid and extremely offensive and contains broken down lung tissue.

In hydatid cysts of the lung the general symptoms are milder than those of abscess. The percussion note, in the center of the affected area, has a more absolute flatness. The characteristic hooklets in the expectorated matter are decisive when obtained.

Abscess of the liver, sub-phrenic abscess, mediastinal abscess, encysted empyema, etc., breaking into the lung, may usually be disgnosed through the history and physical signs present prior to the involvement of the lung. Pus, from these sources, discharged through the bronchi, will not contain fibers of pulmonary tissue except, perhaps, at the immediate time of rupture.

Abscess and empyema are alike most frequent after pneumonia, and if the latter be encysted, as is likely in a pneumococcus infection, it may readily be mistaken for abscess, especially if it discharges into a bronchus. The constitutional disturbance attending abscess is more severe than with empyema, also, the fever, rigors, sweating, pain and cough are more severe. Empyema shows more change in the contour of the affected side and more limitation of motion. Loss of fremitus, less abrupt transition from flatness to partial resonance, greater interference with conduction of voice and respiratory sounds also characterize empyema. When an empyema discharges through a bronchus the signs may resemble those of a cavity in the lung so closely as to make the distinction between them impossible by the physical signs alone.

The following case illustrates this :

Little girl aged 6 years. Had an indefinite illness lasting three months with a history of irregular fever. At time of examination had a temperature range of from 99°F. to 102.5° F. There were signs of effusion in the left pleura. Drainage

was advised but was delayed for some time. A small amount of pus was discharged through an opening between the fourth and fifth ribs. A few weeks after this the child began to spit up pus and the temperature range was from 100° F. to 105° F. There were now signs of a cavity under the center of the left scapula. The lower left lung was much retracted. The physical signs were identical with those of a cavity in the lung substance. Examination of the discharge showed at first pneumococci and streptococci; later a few tubercle bacilli.

TREATMENT.—The medical treatment of abscess of the lung consists in nourishing food and the liberal use of such tonics as iron, quinine and strychnia; the employment of such antiseptic inhalations as have been recommended in bronchiectasis, if the cavity has emptied and the discharge is fetid. Pyæmic abscesses are usually beyond treatment, medical or surgical.

In single abscess following pneumonia, if the discharge is free and septic symptoms not alarming, it is well to postpone surgical interference in order to see if the cavity will not close by cicatrization. In deciding the advisability of operation the points to be considered are the general condition of the patient, the nature of the primary disease, the location of the cavity, whether it is multiple or single abscess, the possibility of free drainage and the condition of the pleural tissues. Cases of pulmonary collapse, circumscribed gangrene and abscess resulting from septic accumulations in the bronchi from the pressure of tumors, aneurisms, injuries to the throat, etc., are not surgical cases and these conditions should always be excluded.

After having located the diseased area as nearly as possible, the presence of pus must be determined by exploratory puncture. These punctures may be repeated several times if necessary to locate the cavity. It is not always easy to locate a cavity with the exploring needle, as the location is apt to be higher in the lung than the signs indicate. The exploration may be made without anæsthesia, though in many cases this is impossible. Chloroform is the best anæsthetic for this purpose and the anæsthesia must not be profound. If pus

is obtained the needle is left in place as a guide, and resection
of one or more ribs is proceeded with. As it is difficult to tell
how much room is needed, it is well to make a vertical incision
instead of the oblique one often used in operating for
empyema.

As it is often impossible to tell with certainty that pleural
adhesions are present it is well to proceed, up to the point of
incising the pleura, as if they were absent. Godlee recom-
mends stitching the pleural surfaces together, including a space
from one and a half to two inches in diameter, with six or
eight stitches. The pleura is now incised and if air draws
through into the pleural cavity, more stitches are introduced.
This method has several advantages over a primary operation
for producing pleural adhesions. If the abscess is near the
surface of the lung a pair of sharp-pointed forceps are intro-
duced along side of the needle, the latter is withdrawn and the
opening enlarged with the finger. Even in deep-seated ab-
scesses it is probable that this method of entering the lung is
preferable to the use of the actual cautery. Hæmorrhage may
be controlled by plugging. The drainage tube should be fixed
to the chest wall with plaster, as it is more likely to become
displaced than in empyema. The wound of the soft tissues
should be treated with chloride of zinc (40 grains to the
ounce) and packed, around the tube, with gauze wet in the
same solution and covered with iodoform (Godlee).

Irrigation of the cavity should not be practiced. Anti-
septic powders may be introduced, but they do little good. A
few layers of gauze covered by oakum or wood wool is a con-
venient dressing when the discharge is abundant and offensive.
Frequent change of dressings is necessary. The tube should
be removed frequently and cleaned. It should not, however,
be removed for the first two days or it may be difficult to rein-
troduce it. The tube should remain until expectoration has
ceased and the discharge through the wound has diminished
to a teaspoonful or so daily.

PULMONARY GANGRENE.

Gangrene of the lungs is a condition of necrosis and decomposition of lung tissue. It is almost always secondary to inflammatory conditions of the lung. Laennec's division of circumscribed, and diffuse gangrene, is still adopted as most convenient, though in the so-called circumscribed variety the peripheral limitations of the gangrenous area cannot always be defined.

Pulmonary gangrene is not frequent, as the post-mortem statistics of various hospitals show its presence in from only one-half to three-fourths of one per cent. of the cases of pulmonary disease.

ÆTIOLOGY.—The microörganisms which induce gangrene may reach the lungs through the bronchi, blood vessels, or by extension from some adjacent organ. Various organisms are found in the necrotic lung tissue. The staphylococcus pyogenes aureus is supposed to be the primary cause of the necrosis (Bonome). Special microbes have been described, and such organisms as leptothrix, aspergilli, sarcinæ, etc., are frequently found. It is likely that several saprophytic organisms may be involved in the production of gangrene.

Gangrene of the lungs occurs in connection with pneumonia, and while rare its occurrence is favored by lack of nutrition, alcoholism, diabetes, nephritis and senility. Influenza pneumonia may be followed by gangrene. The influence of mixed infections in pneumonia upon the occurrence of gangrene is not fully understood. Lobular pneumonia, secondary to the infectious diseases, may rarely terminate in multiple gangrenous foci.

Pulmonary gangrene may supervene upon septic inflammation occurring in connection with chronic pneumonia. Bronchiectasis may cause gangrene by the retention of septic products in the bronchi, or through the occurrence of septic lobular pneumonia of adjacent lung tissue or even of the opposite lung through inhalation of septic material.

Loss of sensibility or paralysis of the larynx allowing

of the passage of food, secretions, etc., into the bronchi may, particularly in the insane, be a factor in producing gangrene of the lungs. Regurgitated matter from the stomach may also be drawn into the lungs. Gangrene of the lungs may occur secondary to cancrum oris, epithelioma of the mouth, tongue or œsophagus, diphtheria, etc. Suppurating bronchial glands may, by establishing communication between the œsophagus and bronchi, cause gangrene. Tumors or aneurisms in the mediastinum or lungs may cause gangrene by obstructing the bronchi and causing dilatation of the tubes, retention of secretions and septic pneumonia and gangrene. Pressure of tumors upon the vessels or nerves of the lungs has been considered a cause of gangrene. Pulmonary tuberculosis rarely results in gangrene.

Septic emboli in the lung may cause gangrene. They may occur from thrombosis of the lateral sinus, thrombi in the uterine veins in puerperal fever, or from the veins surrounding foci of suppuration. Typhoid fever, erysipelas and many other conditions may cause embolic gangrene of the lungs.

Abscess of the liver, fecal abscess, sub-diaphragmatic abscess, cancer or gangrene of the œsophagus may cause gangrene of the lungs by extension. Wounds and injuries of the lungs with extensive extravasation of blood may be followed by gangrene with or without pneumonia. Poisoning by foul air may result in gangrene of the lungs.

MORBID ANATOMY.—If the gangrenous area is near the surface of the lung the pleura will be inflamed and covered with lymph. The gangrenous area may be small or may involve the greater part of a lobe. Embolic gangrene may be wedge-shaped.

In circumscribed gangrene the necrotic area is surrounded by a zone of inflammation by which nature attempts to limit the extension of the process. The gangrenous area may be black, gray or greenish and the necrotic tissue may have been partially expectorated. The contents of the cavity will be fetid, of a pulpy or semi liquid consistence, and may consist largely of blood. The walls of the cavity may be ragged, or may consist of granulation tissue. Well developed

induration may surround the cavity in cases which have lasted some time. The bronchi and vessels of the affected area may remain as shreds in the necrosed mass, or may have been destroyed. The neighboring bronchi are inflamed and contain fetid material.

In circumscribed gangrene there may be one or more foci. The lower lobe is more often affected than the upper lobe, and the peripheral portion of the lung is more involved than the centre. The lung tissue surrounding the gangrenous area is congested or consolidated, and surrounding this is an area of well marked oedema.

When the gangrenous area extends rapidly, severe hæmorrhage may occur from rupture of vessels. Perforation of the pleura may occur. Abscess of the brain is sometimes associated with circumscribed gangrene of the lungs. The bronchitis which accompanies gangrene and which results from the irritation of decomposing material in the bronchi may be very severe.

Diffuse gangrene has no limiting zone of inflammation. The affected area usually has a fetid odor, is gray or black in color, of a doughy or pulpy consistence, or may be a semi-fluid mass in a ragged cavity. The pleura may be necrotic and empyema or pyopneumothorax may be present.

Diffuse gangrene, while rare, is sometimes met with in connection with pneumonia, and rarely after obliteration of a large pulmonary artery though the infection resulting from this cause does not often sphacelate. The larger portion of a lobe may be involved.

CLINICAL HISTORY.—There may be no history indicative of gangrene of the lungs. In the insane or in diabetic subjects and in embolic gangrene even the odor of the sputum and breath may be absent.

The very great variety of ætiological factors for gangrene of the lungs lead us to expect a marked want of uniformity in its clinical history. There may be a history of chills with irregular fever and great prostration. Pain may be present if the pleura is involved. Hæmoptysis may be an early symptom and may rarely be profuse and fatal. Dyspnœa may be severe in cases showing great prostration. Cough may or

may not be troublesome. Fetid breath usually precedes expectoration. The expectoration is yellow, brown or black; purulent or bloody; alkaline at first, but later acid. It forms three layers on standing: an upper of grayish froth; a middle, watery and clear; and a lower layer containing shreds of lung tissue and, possibly, elastic fibers.

In rare instances large fragments of lung tissue may be coughed up. According to Osler elastic tissue is almost invariably present in the sputum. The fetor of the sputum is more marked than in fetid bronchitis or pulmonary abscess. It is particularly offensive when there is free communication between gangrenous cavities and the bronchi.

When gangrene follows pneumonia there may be early prostration, small, feeble, irregular pulse, septic fever and fetid expectoration. In some cases of gangrene after pneumonia, with great prostration, cough and expectoration may be absent.

SYMPTOMS AND DIAGNOSIS.—Fever, cough, pain, dyspnœa, great prostration, fetid breath with expectoration presenting the characteristics of gangrenous sputum are the chief symptoms of gangrene of the lungs. . The physical signs are often more inconclusive than the general symptoms.

Signs of consolidation followed by evidence of the formation of a cavity, especially after pneumonia, and with the characteristic sputum and breath will enable us to diagnose circumscribed gangrene. In children there may be only the signs of broncho-pneumonia.

In diffuse gangrene there will be evidence of more or less complete consolidation of the lung. The voice and respiratory signs of consolidation are modified or wanting as the bronchi of the affected area are more or less occluded. The diagnosis rests mainly on the history, prostration, fever and characteristic sputum and breath. The sputum should be examined microscopically for lung tissue.

Bronchiectasis, putrid bronchitis, pulmonary abscess with fetor, rupture of an empyema into the lung, and local necrosis of a phthisical cavity may resemble pulmonary gangrene. In all lung cavities, other than gangrenous, presenting fetor, the appearance of the fetor is subsequent to the signs of a cavity,

while in gangrene the fetid expectoration precedes the signs of a cavity. In empyema there will be a history of pleurisy prior to the expectoration of a quantity of pus, possibly fetid; but gangrenous odor will usually be wanting and lung tissue will be absent from the expectoration unless at the immediate time of rupture.

TREATMENT.—The treatment of pulmonary gangrene is chiefly supporting, with absolute rest in bed. If there is much pain or cough small doses of morphia may be given, but care should be taken not to depress the patient.

Quinine, strychnia, whisky and milk with concentrated, nourishing food are the chief necessities. Disinfection of the air passages may be accomplished by the methods recommended for bronchiectasis. If the creosote bath cannot be tolerated, inhalations of vapor of carbolic acid ($\frac{3}{4}$ per cent.) may be used. Carbolic acid is also recommended for internal use in doses of from four to twelve grains daily. An inhaler may be used for vaporizing the air passages, saturating the sponge with a solution of creosote, menthol, aristol, eucalyptus, etc., using from ten to fifteen per cent. of any combination desired.

The subcutaneous injection of guaiacol in sterilized oil, in daily doses of fifteen grains or more, is highly recommended by Weil. Several observers have reported good results from these injections. Injections of antiseptic substances directly into the affected area of lung has been advised but is of doubtful utility. Traube advises the internal administration of the acetate of lead; recoveries have been reported under this treatment.

When gangrenous cavities are circumscribed and accessible, drainage is to be considered.

CHAPTER XIV.

PULMONARY TUBERCULOSIS.

Pulmonary tuberculosis (consumption, phthisis) is an infectious disease of the lungs caused by the bacillus tuberculosis. It is the most widespread and prevalent disease affecting the human family. Existing from the earliest times and recognized by Hippocrates it has continued its ravages through centuries and still maintains its position as the most fatal malady of mankind.

One-seventh of all deaths from disease are said to be due to some form of tuberculosis. The U. S. Census Report for 1890 gives 102,188 deaths from consumption. Vaughn estimates, from the Census Report, that one person out of every sixty of the population is affected with tuberculosis.

Statistical evidence is in favor of the decrease of tuberculosis, and some local statistics show marked diminution of the disease. According to Russell there was a decrease of 44 per cent. in the death-rate from tuberculosis in the city of Glasgow from 1860 to 1894. In Massachusetts there is reported a decline of 20.2 per cent. per 10,000 in the death-rate from pulmonary tuberculosis between 1853 and 1895. Biggs, commenting on the prevention of tuberculosis by the New York Department of Health, says, "The death-rate in New York from tuberculosis shows an almost regular and rather rapid decrease from year to year." Pollock says, "We have witnessed the immense decrease of deaths from phthisis and a decided lengthening of its duration. Fewer die of it, and are slow to die when affected."

The modern conception of the nature of tuberculosis, the result of the discoveries of Koch, is a striking endorsement of the doctrines of Laennec who maintained the unity of tuberculous processes; also of Villemin who in 1865 demonstrated the infective nature of tuberculosis by inoculation experiments.

ÆTIOLOGY.—We will not consider here the morphological characteristics of the tubercle bacillus nor the conditions governing its culture and existence. In the vast majority of instances its presence in the lungs is the result of post natal infection. This infection occurs through the air passages or through the blood vessels, and, in rare instances, by extension from the bronchial lymphatics. Infection may occur in several ways.

Hereditary transmission. The possibility of inherited tuberculosis has long been a matter of dispute between those who believe in its direct inheritance (Cohnheim, Baumgarten) and those who believe only in the inheritance of constitutional predisposing factors. Sims Woodhead says, "From the public health point of view the tubercle bacillus must be looked upon as the *fons et origo mali,* and all statistics that have recently been collected go to prove that the tubercle bacillus does its work, as a rule, in the post natal period. Although there may be a few cases of congenital tuberculosis, all statistics point to the fact that tuberculosis is contracted after birth." According to Ransome heredity may act through direct, active infection before birth; through hereditary transmission of latent germs; and through the transmission of a susceptible constitution, but heredity has much less to do with consumption than is popularly supposed.

Direct transmission must occur through the sperm, the ovum or through the blood by the medium of the placenta. Although bacilli have been found in the semen (Jani, Weigert) experimental results (Gartner) and clinical evidence are against this method of transmission. Osler considers the fact that the bacillus will not attack the nuclear material of which the spermatozoön is made as good negative evidence on this line. Baumgarten has detected bacilli in the ovary of the rabbit artificially fecundated with tuberculous semen. The possibility of infection by the ovum cannot be denied in view of the above experimental facts.

The most frequent method of congenital transmission is through the blood current by way of the placenta. This was suggested by Cohnheim and developed by Baumgarten, who believes the bacillus is introduced through the placenta and

umbilical vein ("placental infection"), or by the ovum itself ("germinative or congenital infection"). Buggs has found bacilli in the uterine wall at the site of the placental insertion, in the blood of the umbilical vein, and in the vessels of the infant's liver. Against Virchow's objection that the bacilli, in germinative infection, would interfere with the development of the ovum, Baumgarten brings his theroy of the latency of tuberculosis. While the possibility of the latency of tuberculosis is borne out by the experiments of Mafucci with avian tuberculosis, the acceptance of Baumgarten's application of his theory in explanation of atavism in tuberculosis is hardly to be expected.

Both clinical evidence—as shown by the presence of tuberculosis in 27.8 per cent. in 2,576 autopsies on children who died during the first year of life (Botz), and by the frequent occurrence of localized tuberculosis in children—and experimental results are in favor of the occurrence of congenital tuberculosis. On the other hand, inoculations made from the tissues of fœtuses from tuberculous mothers are usually negative, and the percentage of cases of congenital tuberculosis is very small. Ogle's statistics show that between the second and tenth years of life there is a great decline in the mortality from tuberculosis, and that between the tenth and twentieth years of life the liability of females to tuberculosis is fifty per cent. greater than males. These facts do not conform with Baumgarten's theory of latent tuberculosis. These matters refer particularly to the general infection of tuberculosis and we will not pursue them further.

The manifestations of parental heredity have been variously estimated at from ten to sixty-six per cent. The frequency of its manifestation is the principal source of the general belief in the heredity of tuberculosis. The belief that fathers transmit the disease most often to sons, and, *per contra,* mothers to daughters, is not borne out by statistics. It is generally stated that females are the subjects of hereditary tuberculosis more often than males, and that heredity is most often from the maternal side.

Infection by inhalation. The inhalation of dust containing bacilli is probably the most frequent method of infection.

Flügge thinks that infection is not produced by dried sputum or dust containing bacilli, but by finely divided particles of sputum floating in the atmosphere.

The greater frequency of tuberculosis in towns and cities and in the most condensed districts of these places indicate the greater facilities for infection which attend the massing of many individuals within restricted limits. Bacilli have been found in the dust of street-cars and various public places. Nutall estimates that a patient with moderately advanced tuberculosis expectorates from one and a half to four billions of bacilli in twenty-four hours. If this be anywhere near the truth the effect of the free exercise of the great American prerogative of frequent and indiscriminate expectoration can be readily surmised. Cornet's inoculation experiments with dust from hospital wards, asylums, prisons, etc., show a degree of infectiousness more or less proportionate to the constancy of occupancy by tuberculous cases, and of inverse relation to the degree of cleanliness, etc. Medical wards were six times as infective as surgical wards. Again, bacilli were not found in dust from two wards occupied by tuberculous cases. Pollock, judging from the results in the Brompton Hospital for Consumptives, does not favor the idea that hospital infection is frequent. Hance found the dust from various public places infective. Joccoud, Dumontpallier think contagion in hospitals unlikely, Pean thinks it likely. Terrier, Debove think hospital contagion extremely likely to occur.

The fact that the vast majority of cases of tuberculosis are of the respiratory system; the great prevalence of the disease in institutions where inmates are confined to close quarters and a limited amount of fresh air; and the direct relation between the incidence of the disease and the degree of intimacy of association with tuberculous subjects or infected dwellings or places, all emphasize the possibilities of infection by inhalation.

Infection by inoculation. The experiments of Villemin, Cohnheim, and Salmonson have proved the possibility of this mode of infection. Inoculation may occur through the skin by washing soiled linen, scratches from a broken spittoon, infected earrings, infected hypodermic syringe, in making post

mortem examinations on tuberculous subjects, and in perform-
ing circumcision.   Baumgarten says that inoculation of the
superficial layers of the skin is impossible, subcutaneous inocu-
lation being necessary to insure infection.   Inoculation by the
alimentary canal may occur through kissing consumptives, by
the use of infected tableware, and by contact with coughed
up particles of sputum.   Direct inoculation through the gen-
erative passages, while possible, must be extremely rare.   Ger-
lach, Ballinger and others have shown the infective quality of
milk from tuberculous cattle.   [Hay found bacilli in 51 out
of 351 separate samples of cows' milk, and in 4 out of 204
mixed samples.]   Ernst has shown that milk may be infective
even in the absence of tuberculous mammitis in the animal
from which it is obtained.   According to Bang butter made
from the milk of tuberculous cows may be infective.   Osler
thinks the frequency of mesenteric tuberculosis in children
may be explained by the infective qualities of milk.

Experiments show the possibility of infection in animals
by the use of the flesh of tuberculous subjects.   This mode of
infection is extremely rare in human tuberculosis but presents
possibilities which should be guarded against.   The Royal
Commission upon Tuberculosis states, "We must believe that
any person who takes tuberculous matter into his body incurs
some risk of acquiring tuberculous disease."

Inoculation, on the whole, plays an unimportant part in
the mode of infection of human tuberculosis.

Predisposing causes of infection.   The geographical dis-
tribution of pulmonary tuberculosis shows that while prac-
tically absent in certain arctic regions, deserts, and at great
altitudes, yet in no habitable part of the earth does there exist
complete immunity.

Climate.   The climatic conditions favoring infection are
moisture and heat.   The bad effect of wet sub-soil was shown
by Bowditch in 1862, who said "A residence on or near damp
soil is one of the primal causes of consumption in Massachu-
setts."   Buchanan's investigations corroborate this view in
regard to the effect of wetness of soil.   While hot climates
seem to favor an active course for tuberculosis, we may accept
Hirsch's statement that "The mean level of the temperature

has no significance for the frequency or variety of phthisis in any locality." Low altitudes, moisture, rapid and extensive changes in temperature, and cloudiness are the climatic features which favor pulmonary tuberculosis.

Race. No race is exempt. Aboriginal people develop susceptibility when removed from their usual conditions of existence. Thus the Negro in Africa is not as susceptible as in America though this race exhibits unusual susceptibility under all conditions. Susceptibility in the American Indian has increased since the conditions governing his early history have changed. Statistics show that a relative immunity exists among the Jews.

Sex. Apart from difference at certain ages there is practically no variation between the sexes as to liability to infection, the mean annual mortality for a period of thirty years being 2,814 for males, and 2,428 for females (Kidd). Ogle's statistics show that for the first few years of life the mortality for the two sexes is alike; between five and thirty-five the female mortality greatly exceeds the male; after this period the male mortality exceeds the female. This applies to pulmonary tuberculosis and not to general tuberculosis. The influence of pregnancy on infection has been much discussed. Pollock states that the periods of puberty, of gestation, of parturition and lactation are dangerous to individuals predisposed to tuberculosis. This is true with possibly the exception of pregnancy in certain individuals in whom the latter state stimulates nutrition to an extent which antagonizes all pulmonary diseases.

Age. No age is exempt. Hippocrates states the recognized fact that pulmonary tuberculosis is most frequent from the eighteenth to the thirty-fifth year. [1881—1890, of 397,-559 total deaths, 118,598 were from consumption, at ages between 25 and 35 years—Ransome.] Landousy has demonstrated the tuberculous nature of several cases of bronchopneumonia in children under two years of age.

Occupation and environment. Occupations involving the breathing of impure, overheated, or dusty air predispose to infection. Sedentary or indoor work, bad ventilation and drainage, the overcrowding of employes in close quarters

etc., are factors of environment. Flick's investigations in Philadelphia, and Ransome's in Manchester, England, have demonstrated that infection clings to certain dwellings once they have become infected by tuberculous residents.

Individual predisposition. The modern understanding of the infectious nature of tuberculosis has tended to modify the ancient theory of predisposition—a theory dating from the time of Hippocrates and Galen and postulated on presence of a *special* predisposition to tuberculosis. It is argued by many that individual predisposition consists merely in an inherited general constitutional liability to all depressing influences, i. e., the predisposing factors of tuberculosis. This argument does not materially affect the position of those who hold to the doctrine of predisposition.

Statistics undoubtedly show family tendency to tuberculosis, and, moreover, show that the mother's influence is greater than the father's as regards both sexes; and that the disease appears at an earlier age when there is family tendency, particularly in females. Could Baumgarten's theory of latent tuberculosis be established in all its phases the matter of predisposition would assume a new position.

General and local diseases. Measles, whooping-cough, influenza, variola, diabetes, insanity, syphilis, and, in some degree, all depressing diseases predispose to tuberculosis. Bronchitis, pneumonia (rarely), pleurisy, chronic heart disease particularly stenosis of the pulmonic valves, are predisposing factors to tuberculosis of the lungs.

The opinion that a large proportion of pleurisies are tuberculous is supported by post-mortem evidence and by the subsequent history of these cases. Rokitansky taught an antagonism between tuberculosis of the lungs and physical changes in the heart resulting in obstruction to the pulmonary circulation. This has been denied by many, but is supported by several observers of great clinical experience. Louis believed in the antagonism between mitral disease and tuberculosis, and J. E. Graham states that mitral stenosis antagonizes tuberculosis of the lungs. Ott believes that left-sided disease of the heart is antagonistic to pulmonary tuberculosis. The fact that mitral disease is occasionally associated with tuber-

culosis of the lung does not invalidate these views. My own experience is decidedly in accord with these observers as I have never seen tuberculosis of the lung develop in a person the subject of disease of the left side of the heart which particularly affected the pulmonary circulation.

The following case is of interest in this connection:

Woman aged 24. Married. A few weeks after childbirth she developed an acute, lobular, tubercular pneumonia of the right lung. After three weeks of severe illness there were two weeks of improvement when another attack of pneumonia developed, followed by signs of a cavity in the right lung. Shortly afterwards she developed an acute rheumatism with signs of an acute endocarditis with mitral localization. After the appearance of the mitral lesion (regurgitant) there was much improvement in the lung symptoms and the patient was soon up and about, and after several months adopted a change of climate. While recovery was impossible in this case the advancement of the disease was undoubtedly checked by the occurrence of the mitral lesion.

It is claimed that traumatism is a factor in producing tuberculosis. Surgeons, generally, recognize the effect of traumatism in disseminating local tuberculosis. Mendelssohn has recorded cases where contusions of the chest have been followed by pulmonary tuberculosis.

MORBID ANATOMY.—Histologically a tuberculous nodule is a non-vascular structure consisting of a central substance made up of one or more multinucleated giant cells, or of a granular substance surrounded by giant cells. These cells may be connected by a network of anastomotic processes; outside of the central structure there is usually found large epithelioid cells with large nuclei and granular protoplasm. These cells lie in the network formed by the processes from the giant cells; surrounding these structures is a more or less indefinite zone of lymphoid cells contained in a homogeneous, or fibrillated reticulum which is best marked in slowly developed lesions.

The changes which occur in a tuberculous nodule are

caseation and fibrosis. Whether the change assumes one form or the other depends on the mode and extent of the infection, and on the resisting power of the individual. This defensive action of the body against the action of the bacilli is supposed to depend in part on phagocytosis (Metschnikoff), and partly on an antitoxic action of the serum and blood plasma which is derived from the leucocytes either by a process of excretion or by their destruction.

In caseation the cells in the center of the nodule become swollen, fused, and lose their structure (coagulation necrosis—Cohnheim) becoming opaque and homogeneous (gray tubercle); fatty degeneration occurs and the nodule becomes cheesy and of soft consistence and yellow color (yellow tubercle), particularly in large and diffused lesions. Caseous lesions may soften and form a cavity or they may become calcified by the deposit of lime salts and be expectorated or encapsulated.

Fibrosis occurs when coagulation necrosis is limited and the zone of lymphoid tissue is transformed into dense contracting tissue. This change postulates resisting power of the tissues and is Nature's method of limiting the lesion. It occurs in infections of limited severity and extent especially when occurring through the vessels.

The type of the lesion in the lung will depend on the mode of infection. If this is through the vessels, coming from a caseous focus in some other organ or tissue (Buhl) or in the lung itself or as part of a general tuberculosis, there results a more or less extensive eruption of disseminated miliary nodules from embolic arrest in the alveolar walls. These nodules are most numerous at or near the apex of the lung. The lung tissue between the nodules may be normal, congested or œdematous, or may present areas of consolidation. In some cases there is a tendency to necrosis and softening of the nodules if the case is not too rapidly fatal; in others there is a tendency to fibroid induration of the alveolar walls and surrounding tissues. Fusion of the nodules, in the earlier stages before the softening of "yellow" tubercle appears may give the tissue a gray, gelatinous appearance (Laennec's gray infiltration).

If the infection occurs through the air passages lodgment is found in the terminal bronchioles or the alveoli, and there results peri-bronchitis and broncho-pneumonia. The structural changes are, chiefly: epithelial accumulation within the alveoli; fibrinous exudation and leucocytes in the alveoli; cellular infiltration and thickening of the alveolar walls and terminal bronchi; increase in the interlobular connective tissue. These changes are variously associated and the preponderance of one or the other will depend on the type and stage of the lesion. The alveolar accumulation of epithelial cells is similar to that of ordinary lobular pneumonia and is a characteristic lesion of the majority of cases of acute tuberculosis of the lungs, and of the acute phases of the so-called fibro-caseous variety.

Fibrinous exudation and leucocytes within the alveoli are found in some of the most acute forms of the disease—the lobar type of pneumonic tuberculosis. The consolidation resembles that of croupous pneumonia but there is more or less epithelial proliferation. These intra-alveolar changes give the tissue a reddish or grayish-yellow color. It is friable and soft with a more or less lobulated periphery. The breaking down of the tissue may form cavities of various sizes with the soft, irregular walls.

Infiltration and thickening of the alveolar walls and bronchioles is a constant and characteristic lesion of all cases of pulmonary tuberculosis. It replaces and gradually obliterates both the alveoli and the pulmonary capillaries, which latter effect is probably largely responsible for the tendency to retrograde metamorphosis exhibited by fibrocaseous tuberculosis of which this lesion is particularly characteristic.

Increase in the interlobular connective tissue is a feature of the chronic forms of tuberculosis. There is a greater tendency to fibroid development than in the inter-alveolar tissue, less tendency to degeneration, and blood supply is generally maintained. Extensive induration of the lung may result from this process in chronic tuberculosis. Cavities, when present, have dense, fibrous walls.

More or less bronchial catarrh is associated with these changes. The bronchial wall is infiltrated with cells and ulcer-

ation may be present. The peri-bronchial tissue may show cell infiltration. Large cavities may be traversed by fibrous bands formed from bronchi, or arterial chords which are generally closed by thrombosis and obliterating changes before ulceration of the wall results in hæmorrhage. Aneurism of the vessel may occur, however, before these changes take place, and be followed by fatal hæmorrhage.

Bronchial stenosis, dilatation, ulceration; pulmonary emphysema, collapse, œdema, gangrene; pleural adhesions, thickening; miliary tuberculosis of the pleura or pericardium; pneumothorax, pyopneumothorax, and tubercular enlargement of the bronchial glands in children (Loomis, Ballinger) are the principal associated chest lesions. Lesions of other organs are frequent.

The lung lesions occur in endless variety and degrees, and we will not attempt to recount them. Miliary nodules are frequently found in the lung having been distributed by the circulation from a focus of inspiratory origin and pneumonic nature. More frequently the secondary foci are due to transmission of infective material by aspiration aided by gravitation from one part of the lung to another.

The characteristic apical invasion of tuberculosis which is common to all varieties of the disease is generally explained on the ground of lessened functional activity. This localization is most commonly an inch or two below the immediate apex and nearer the posterior surface than the anterior. A less common location is just below the outer third of the clavicle in the outer portion of the upper lobe. Fowler shows that tuberculous lesions of the lung progress, as a rule, by distinct routes and almost always from above downward. From the primary foci in the apex, extension is downward in the anterior portion of the lobe just within the margin. The middle lobe of the right lung is usually affected secondarily to the upper right lobe, and late in the disease. It frequently escapes altogether.

The lower lobe of the lung previously affected becomes involved early, usually before necrosis of the upper lobe has occurred and before the opposite apex is affected. The site of the lesion in the lower lobe is a spot midway between the

border of the scapula and the spinous processes of the verte-
bræ and opposite the fifth dorsal spine. Fowler considers the
involvement of this area early in the disease as characteristic
of the chronic varieties of tuberculosis and of immense clini-
cal importance. Extension from this area is backward and
along the line of the interlobular septum. Extension toward
the base is usually by scattered areas. Basic lesions óccurring
in the absence of apical lesion are either non-tubercular, or
the tubercle is secondary. Invasion of the opposite apex, to
that primarily affected, may be early, but usually not till after
the invasion of the lower lobe of the lung primarily affected.
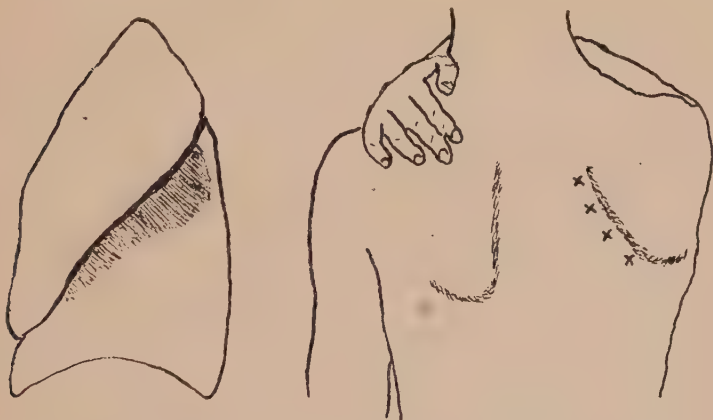
Extension of lesion   Position of arm when vertebral border of
of lower lobe along the   scapula marks line of extension of lesion
interlobar septum.   along interlobar septum.
            (After Fowler.)

The lesion is symmetrical with that of the primary lesion, but
occasionally may affect the upper lobe close to the septum and
opposite the axillary region. Occasionally the lesion crosses
from the upper lobe of one lung to the upper portion of the
opposite lower lobe.

Cavities result from necrosis and ulceration of a tuber-
culous area, usually from the result of these processes upon
the bronchi of the affected area which become dilated from
retained secretion. The center of a caseous area may ulcerate
and form a cavity without involving the bronchi. Three
forms of cavities are found: The recent ulcerative cavity of

acute cases, with soft necrotic walls without limiting membrane,—these cavities may rupture into the pleural cavity and cause pneumothorax: Cavities with well defined walls of limiting membrane which secretes pus and which gradually undergoes slow necrosis with increasing size of the cavity,—these cavities may be crossed by trabeculæ of arterial and bronchial cords, and are found in the chronic varieties of tuberculosis; Stationary cavities, quite small, with smooth lining membrane surrounded by dense, fibrous tissue,—they may communicate with the bronchi (*cicatrices fistuleuses*— Laennec).

CLINICAL HISTORY.—No one infection of the human organism exhibits such a varied clinical history and course as tuberculosis, and especially so in regard to its pulmonary manifestations of which many classifications embodying much variety of nomenclature have been made. It is unnecessary to discuss these classifications.

Basing our distinction partly on the mode of infection and partly on the gross character of the pathological changes, we recognize four more or less distinct types of pulmonary tuberculosis, i. e., miliary tuberculosis, fibroid tuberculosis, acute pneumonic tuberculosis, and chronic pneumonic tuberculosis. The frequent combination of these types of the disease gives pathological and clinical justification for the various classifications which have been alluded to, but for the sake of brevity and clearness the above division has advantages.

Miliary tuberculosis of the lungs occurs as a feature of general tuberculosis, or an eruption of miliary tubercles may occur as a current or terminal event in other forms of pulmonary tuberculosis. A caseous focus may discharge into a branch of the pulmonary vein (Weigert). In some cases, as in those which follow measles, whooping-cough, typhoid fever, etc., no source of infection can be discovered.

Miliary tuberculosis is more common in children than in adults, and is rare after fifty, though it may occur at any age. The lungs are large, hyperæmic and full of discrete tubercles of recent formation about the size of a pin's head and gray in color or semitransparent. When miliary tuberculosis is

secondary to chronic lesion of the lung the tubercles are less clearly defined, are grouped together, show caseation, and are most plentiful in proximity to the original lesion. The primary lesion may assume a broncho-pneumonic type.

There is a history of sudden onset with general malaise, loss of appetite, headache and fever. Hæmoptysis may mark the onset or there may be marked bronchitis with muco-purulent or rusty sputum. Dyspnœa is severe and out of proportion to the physical signs. Cyanosis is a distinctive feature because of its severity. It is most marked in the lips and finger tips. The cheeks are flushed, cough troublesome, pulse rapid and feeble, and the respiration may be from forty to eighty per minute. The temperature ranges from 100° to 103.5° F. and according to Fowler presents a continuous and an inverse type. In the former it rises gradually and continues high with slight morning remissions; in the latter the maximum temperature is in the morning instead of the evening.

A typhoid state may develop towards the end of the case. Acute miliary tuberculosis is generally fatal in from two to three weeks though it may last for three or four months.

Fibroid tuberculosis is a chronic miliary tuberculosis occurring most often as a primary affection of the lungs in persons of good resisting power and without predisposition. In these persons a moderately severe or extensive infection through either the vessels or air passages may result in miliary tubercle with a tendency to fibroid transformation rather than caseation and softening.

It occurs most often in men and in those whose dusty occupations favor sclerosis of the lung tissue. Apical invasion is the rule as in other forms of tuberculosis though secondary lesion may occur in any portion of the lung. The lesions occur as isolated nodules which are indurated, pigmented and feel like shot in the lung tissue; or are grouped as nodules or areas of induration. Fibroid miliary tuberculosis of the lung may occur in connection with intestinal and mesenteric tuberculosis, and there is also, as a rule, miliary tuberculosis of the pleural and pericardial surfaces, as in the following case:

Woman aged 57. Family history good, previous health fair. Sick several months with irregular diarrhœa. Had lost flesh rapidly. Temperature 99.5° F., pulse 100. Lungs showed general induration most marked in the apices. No bronchitis, cough or expectoration. The abdomen was flat and irregular, deeply seated nodules could be felt. The diarrhœa could not be controlled for more than a day at a time. She became greatly emaciated and died three months after coming under observation.

Post mortem examination showed intestinal and mesenteric tuberculosis, miliary tuberculosis of the pleural and pericardial surfaces and general disseminated miliary tuberculosis of both lungs with well-marked fibrosis.

The history is one of gradual onset, with some cough and gradual loss of flesh and strength, extending over a period of several years, or, with a history of this kind lasting for a year or two there may be entire absence of all subjective symptoms, the process having been arrested. Again, a portion of the lung—usually the apex—may be markedly changed, with no history of illness other than gradual loss of strength.

More or less cough is present though it may be absent. Fever is usually slight (one-half to one degree). Quite frequently the course of the disease is apyrexial. Hæmoptysis is common in the early stages, may be recurrent, is usually slight, may be profuse, but in either case is not connected with the occurrence of lobular inflammation as it is in the pneumonic type of the disease. Diarrhœa and night sweats are usually absent. In advanced cases with marked retraction of the lung and thickened pleura the cough may be violent, paroxysmal, and tend to produce vomiting. Dyspnœa and rapid pulse may be present in cases where emphysema has developed. Muco-purulent or fetid expectoration may be present and the cases may ultimately present the various clinical features of advanced sclerosis of the lungs.

This form of tuberculosis tends more than any other to undergo arrest, or, at least, to run a prolonged course. It may last from ten to twenty years.

Acute pneumonic tuberculosis (galloping consumption,

phthisis florida, caseous tuberculosis) occurs in two forms: the disseminated lobular or broncho-pneumonic type, and the lobar or pneumonic type. The former occurs most often in children or young people, and in those with a predisposition to tuberculosis. These two types constitute only about two and one-half per cent. of the total cases of pulmonary tuberculosis. The lobar variety is rare and occurs more often in adults than in children.

Acute pneumonic tuberculosis is usually primary though it may occur secondary to chronic pulmonary tuberculosis.

In the lobular type there is acute inflammatory broncho-pneumonia beginning in the smaller bronchi which are filled with cheesy substance while the alveoli are filled with catarrhal products. These areas are first grayish-red and later become white and caseous. By fusion they may resemble the lobar type, but the lobular distribution can usually be made out. These caseous areas, from 1-3 cm. in diameter, may be irregularly scattered throughout the lungs. Usually they are most numerous at the apex. Smaller, opaque, gray or caseous nodules may be present. Small or large cavities may be found at the apex or base of the lungs. Prudden has demonstrated that these changes may occur without the agency of a secondary infection. This form of pneumonia may follow the aspiration of blood into the bronchi in cases of hæmorrhage (tuberculosis aspiration pneumonia—Bäumler).

In the lobar type a single lobe or the entire lung may be involved. The affected area is gray, yellowish-white and caseous. The surface is smooth or granular, uniformly changed, and it may be very difficult to detect any tuberculous foci. Softening and cavities may be present in the apex. In some cases the change is less uniform, there being aggregated masses of caseous areas surrounded by hyperæmic or consolidated tissue.

The onset of the lobular form is variable. Hæmorrhage may be the initial symptom, followed by chills, high temperature, rapid pulse and respiration, and rapid loss of flesh and strength. In children the disease usually follows measles or whooping-cough. While convalescing from these diseases the temperature may rise suddenly, there is cough, dyspnœa, and

signs of consolidation. The case commonly ends fatally in from a few days to two or three weeks, or, with the appearance of hectic fever and sweats the disease may pass into a condition of chronic phthisis.
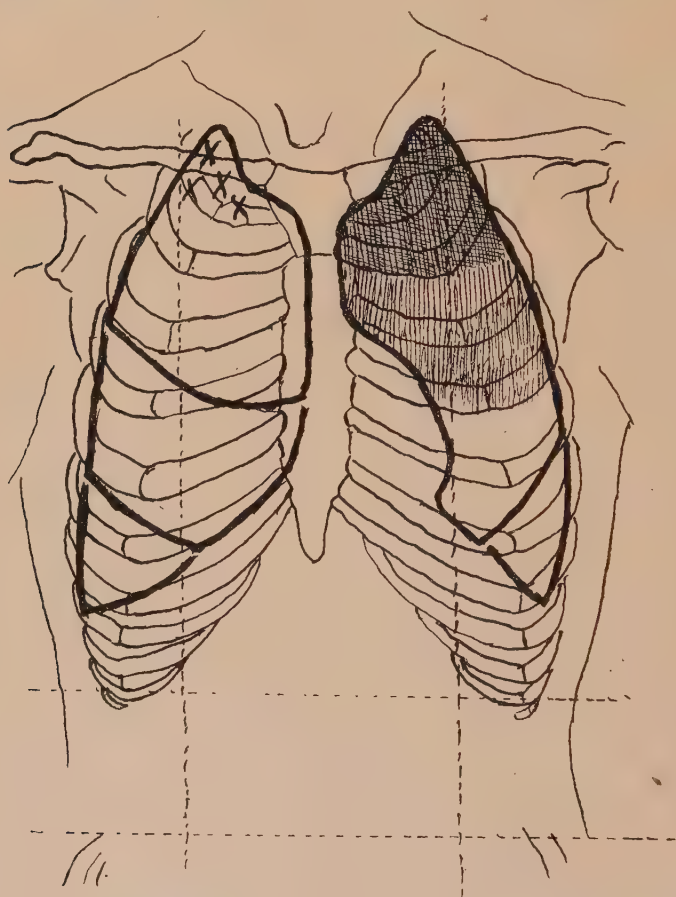
Extension of tuberculous lesion. Dark shade—primary lesion; light shade—extension of primary lesion; X—secondary lesion of opposite apex.

The temperature in the lobular form is high and of a remittent type varying from 101-104° F. In the later stages with diminished vitality the temperature may range from 96 or 97° F. in the morning to 103° F. in the evening (Fowler).

In the lobar type of the disease the onset is sudden. There

is chill, rapid rise in temperature, pain in the side, cough, mucoid or rusty sputum, dyspnœa and, perhaps, cyanosis. In all respects the attack is like an ordinary pneumonia. At the eighth or tenth day, however, the temperature becomes irregular, the pulse rapid, expectoration muco-purulent and, perhaps, greenish (Traube), and sweating may occur. Death may occur in from one to four weeks; the case may be prolonged for two or three months or it may become one of chronic phthisis.

Chronic pneumonic tuberculosis (chronic ulcerative tuberculosis, fibro-caseous tuberculosis). This form of the disease constitutes the great majority of the cases of pulmonary tuberculosis in which caseation and excavation occur. While the lesion is primarily tubercular, in nearly all cases it ultimately becomes a mixed infection, and some of the best known and most troublesome features of the disease are due to secondary infection with pyogenic organisms.

The lesions of this variety of tuberculosis show great variety in nature and distribution. Broncho-pneumonia, miliary tubercles which are disseminated either by the air passages or the vessels, sclerosis, caseation and excavation are the chief changes. The formation of cavities in this disease is favored by the occurrence of secondary infection (Prudden). Tubercular changes in the bronchi, bronchial glands, pleuræ, and in the other organs and tissues of the body are frequent. Tubercular laryngitis is common in this form of the disease.

The onset of chronic pneumonic tuberculosis is very varied. It may come on insidiously, and marked changes may have occurred in the lung before any symptoms develop Again, the same insidious invasion is accompanied only by symptoms of anæmia and gastric disturbance, particularly in young females, or there may be irregular fever simulating malaria. In any of these types if the temperature is watched it will usually be found from one-half to one degree above normal in the afternoon or evening. A persistent temperature of this kind is suspicious of, but does not necessarily indicate, tubercular infection.

In many cases there is a history of repeated attacks of bronchitis which gradually become prolonged, or a winter cough may be protracted through the summer and be accom-

panied by emaciation and night sweats. This is a frequent history following influenza bronchitis occurring during the winter or spring months. Many cases present no history but that of a more or less constant cough which is dry and not troublesome. They will often not acknowledge a cough unless closely questioned.

There is, at times, a history of one or more attacks of
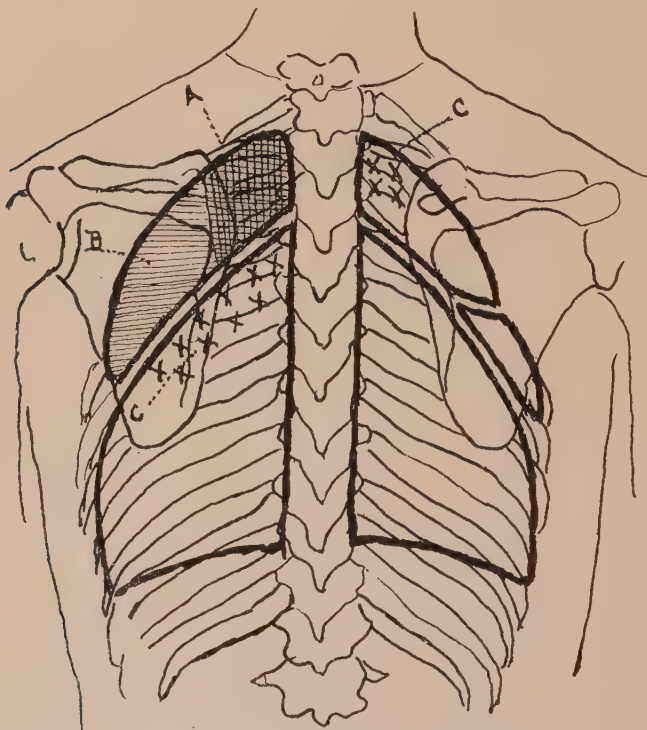
Showing relation of lobes to posterior chest wall, also extension of tuberculous lesion. A—primary lesion; B—extension of primary lesion; C.C—secondary deposits.

pleurisy, either dry or with effusion; the pleurisy has disappeared, but following it there is a history of cough, fever, night sweats and emaciation.

In some instances the larynx is the primary seat of the disease, though usually it is involved secondarily. There may be laryngeal irritation and cough, mucoid expectoration, huskiness and aphonia.

Hæmoptysis may be the first symptom in some cases. There may be marked hæmorrhage followed within a short time by signs of lesion in the lung, or the hæmorrhage may be slight and the interval before lung symptoms develop may be long. In other cases there are repeated slight hæmorrhages. In many cases when any considerable hæmorrhage occurs there is already disease of the lungs. Hæmorrhage is present in from sixty to eighty per cent. of the cases of pulmonary tuberculosis (Osler). In most cases it recurs, and may be a prominent feature (hæmorrhagic phthisis). Stricker's percentages for soldiers are: without special cause, 86.8 per cent. tuberculous; after exercise, 74.4 per cent. tuberculous; after swimming or direct injury to the thorax, one-half not tuberculous. The bleeding is usually sudden, the first intimation the patient has of it is a salty, warm taste in the mouth. The hæmorrhage may come on while coughing but more often does not. The amount is usually small especially in the early history of the case. [In 69 per cent. of 4,125 cases of hæmoptysis in the Brompton Hospital the quantity of blood expectorated was less than one-half of an ounce.]

Cervical or axillary tuberculous adenitis may precede by months or years the development of pulmonary lesion, though in some cases the development of the two conditions is coincident.

Pain is frequently complained of and is usually connected with pleurisy. It is most often felt in the lower and lateral regions of the chest, or beneath the upper portion of the scapula.

Cough is a very variable symptom. It may be dry and hacking at first, later it is loose, with muco-purulent expectoration. It may be paroxysmal if there are cavities. Paroxysmal morning cough may be followed by nausea and vomiting.

The respiration is usually increased, somewhat in proportion to the amount of tissue involved and to the degree of fever. It may be 26-30 per minute during the exacerbation of all symptoms which attend the invasion of new areas of lung tissue by broncho-pneumonia. Dyspnœa is not common even when considerable lung tissue is involved, except in

chronic cases with marked sclerosis and a weak heart. Nervous dyspnœa may be troublesome at times. Cyanosis is rare except where miliary tubercles invade the sound portion of a lung partly crippled by chronic disease.

Fever is an almost constant element of the clinical history of chronic tuberculosis. There are apyrexial periods, and in arrested tuberculosis fever will be absent. As a rule it is a fair indication of the activity of the process in the lungs. Every variety of range is exhibited. In the early stage or in quiescent cases the fever may range from 98 to 99° F. As the disease advances and softening occurs, the fever will range from 99 to 101° F., the morning remission may be as low as 97° F. A sudden and maintained elevation of 102-103° F. is indicative of a new area of lobular inflammation or a new tract of miliary infiltration. Whatever type the temperature may exhibit it may maintain this type for weeks or months at a time if there are no disturbing elements. Towards the termination of a fatal case the temperature may be subnormal for a day or two. The cases exhibiting the most irregular and fluctuating temperature are those in which secondary infection exists. Maragliano, Stern and Erbman believe that hectic fever is due to secondary infection and not to tubercular infection. Fowler dissents strongly from this view.

The pulse is very variable. It is rapid with considerable fever or with extension of the disease. According to Wilson Fox it is influenced more by the patient's strength than by the degree of fever.

The quickest pulse is usually observed in the morning (Smith, Fox). There is no reliable relation between the pulse and the respiration. Nervous influences affect the pulse greatly. In the late stages the pulse is very rapid and of low tension. Pseudo-angina and palpitation are frequent. According to Powell palpitation is most frequent in patients with considerable retraction of the left lung.

Night sweats occur in most cases though some patients escape entirely. They occur towards morning when the fever drops, or at any time when the patient sleeps there may be sweating. Sweating may appear early and be persistent, but its appearance usually coincides with the beginning of soften-

ing. The severest sweating may disappear late in the disease without any reference to treatment.

Emaciation is usually progressive as long as the process in the lung is advancing, though increase in weight may occur while the disease is progressing if the patient's nutrition is forced. As a rule stationary or increased weight is a sign of quiescence of the disease.

Gastric crises may occur, especially in neurotic subjects and insane delusions and hallucinations are sometimes present.

Diarrhœa may occur early from errors in diet. Occurring late in the disease it is indicative of intestinal ulceration.

Constipation may be troublesome at times. Anal fistula usually occurs late in the disease and is much more common in males than in females.

SYMPTOMS AND DIAGNOSIS.—In miliary tuberculosis besides the dyspnœa, cyanosis and peculiar type of fever, we have the physical signs of bronchitis combined with those of a varying degree of empysema, and, perhaps, those of irregular areas of broncho-pneumonia. The percussion note may be irregularly dull at the base; in the anterior regions of the chest it may be hyperresonant. Sibilant, sonorous, and fine mucous and subcrepitant rales may be present. Pleuritic crepitations may occur from local tubercles (Jürgensen). High-pitched, tubular breathing may be heard at the base of the lungs; in front and above the breathing may be low in pitch and expiration may be somewhat prolonged. Daily alteration in the percussion pitch may be due to the re-expansion of collapsed areas (Eustace Smith). Miliary tuberculosis associated with chronic lesion of the lungs may present few recognizable alterations in the physical signs.

The degree of dyspnœa and cyanosis; the temperature range; together with the discrepancy between the physical signs and the general condition of the patient are important aids in diagnosis. Bacilli are often absent from the sputum, and frequently there is no sputum. Choroidal tubercles may be present, and signs of meningeal tuberculosis or other forms of general infection will aid in the diagnosis. When the lung is involved in connection with acute general tuberculosis the case may resemble typhoid fever, but the temperature is less

regular, may assume the inverse type or may be sub-normal in the morning. The Widal test may decide, though a negative result in the first four or five days does not exclude typhoid fever.

Acute bronchiolitis in children and after measles or whooping-cough, may resemble acute miliary tuberculosis of the lung. The temperature range is more irregular in miliary tuberculosis, and in bronchiolitis the correspondence between the dyspnœa and the degree of pulmonary involvement is more evident.

Fibroid tuberculosis. The symptoms of this form of tuberculosis are those of sclerosis of the lungs with which it is clinically identical. In some cases there is an alar or pterygoid conformation of the chest (Galen, Aretæus) characterized by winged scapulæ, oblique ribs, increased length of chest, depressed shoulders and diminished antero-posterior diameter of the thorax. The costal interspaces are depressed and the inferior ones show inspiratory recession. The lateral expansion is diminished, the movement being mainly perpendicular. Depression of the supra and infraclavicular spaces is present and the sternum may be depressed. The heart may be displaced and epigastric pulsation may be present. The vocal fremitus may be increased or diminished, depending on the degree of induration, its proximity to the surface and the degree of emphysema present. Moderate induration and emphysema may give more marked fremitus than a more solid and airless portion of the lung.

The percussion note may be high-pitched and hard, or resonant from emphysema. The respiration is weak with prolonged, high-pitched expiration. Wavy, interrupted or cog-wheeled respiration may be present. Fine, crackling rales may be heard in various portions of the lungs. If cavities are present they are usually dry and present the usual signs which are apt to have an amphoric character. These changes in the lung are most marked in the apices. Retraction of the apex below the upper level of the clavicle with signs of induration is decidedly indicative of tubercular disease. Local changes with collateral emphysema may be difficult to detect.

Repeated examinations of the sputum may fail to show

bacilli, but sooner or later they can usually be found. In primary or secondary basic tuberculosis of a fibroid nature the diagnosis may be difficult, the only positive proof of its nature being the presence of tubercle bacilli. The presence of old lesions in the apices may aid in the diagnosis. In chronic miliary tuberculosis of the lungs associated with mesenteric or intestinal tuberculosis the physical signs may be very indefinite.

Acute pneumonic tuberculosis. In the lobar form the physical signs may be those of ordinary pneumonia, i. e., dullness, bronchial breathing and bronchophony. Pleuritic friction may be present. The breath sounds may be feeble or, at times, absent. The diagnosis may be aided by a more remittent temperature than is common in pneumonia. The absence of crisis; the presence of rapid pulse, sweating, emaciation, muco-purulent expectoration, and, sooner or later, the presence of bacilli in the sputum will establish the diagnosis. The diagnosis must often be necessarily deferred till after the acute stage. Fowler remarks that "These cases are of very rare occurrence, and any one who feared that every case of ordinary pneumonia he observed might prove to be of this nature would by his unusual alertness do more harm than good."

In the lobular or disseminated form of the disease the first signs may be fine, crackling, metallic rales at the apex, just outside of the nipple, or diffuse rales all through the lungs. Local signs of consolidation, particularly breathing of a bronchial character and bronchophony may be heard, but if the consolidated area is not near the surface of the lung these signs are indefinite. Several disseminated areas of consolidation can sometimes be made out. If cavities are present they will manifest the usual signs.

The diagnosis rests on the presence of bacilli in the sputum, or of elastic tissue if the lung is breaking down. Ordinary broncho-pneumonia in children may, when attended with dilation of the tubes, be indistinguishable from tubercular pneumonia. If the sputum cannot be obtained it is well to withhold opinion as to the exact nature of the case.

Chronic pneumonic tuberculosis.—The subjects of this

form of the disease present the pale, loose, dry skin, blue veins, flushed cheeks, pearly conjunctivæ and pale membranes which make up the composite general appearance popularly known as "consumptive-looking," which, taken with the clinical history, is often almost sufficient for a diagnosis.

Cough, which is one of the earliest symptoms, varies greatly in character and constancy. In some cases there may be little or no expectoration. The early history of fibrous, miliary, or caseous tuberculosis without softening is much more likely to show expectoration than is that of chronic pneumonic tuberculosis. When bronchitis precedes the disease the expectoration is likely to be profuse. In the early stages it may consist of viscid, glairy, or watery mucus; later it contains small gray, greenish, purulent masses. If softening has occurred the sputum is muco-purulent and yellow or greenish in color and may contain flattened masses (numular sputum). Sputum of this kind is likely to come from cavities and has been considered characteristic of phthisis, but it may be present from chronic bronchitis. This sputum is heavy and sinks in water, a fact of much concern to some patients. The character of the sputum is changed by hæmorrhage, gangrene, bronchiectasis and various other associated conditions.

The sputum should be examined in every case for tubercle bacilli. They are most apt to be found in the small gray or yellowish-green masses found in the sputum. The examiner will use whichever of the various methods of staining that appears to him to be the most convenient and reliable. There is no constant relation between the number of bacilli and the activity of the process in the lung, nor does the continued presence of bacilli necessarily mean that the disease is progressing actively. As a rule diminution in the amount and purulency of the sputum and in the number of the bacilli indicates a lessened activity of the disease.

Elastic tissue is an important element in phthisical sputum at times. It may come from the bronchi, alveoli, or from the arterial wall. That from the bronchi presents as a long net-work or as three or four long fibres lying close together; that from the alveoli shows bunched fibres with a curled or

alveolar arrangement; that from the arteries may show as a sheet of fibres. In order to examine the fibers place some of the purulent material upon a glass plate about four inches square and press it out into a thin layer with another plate about three inches square. Shown against a black background the elastic tissue appears as grayish-yellow spots which can be removed to an ordinary slide for further examination (Andrew Clark's method). Fenwick's method is to boil equal parts of sputum and a solution of caustic soda (20 grains to the ounce) for a few minutes. Let stand for 24 hours in a conical glass. Examine sediment. The centrifuge will facilitate this process.

The curved and alveolar arrangement of the fibers is the most reliable evidence of destructive processes which are almost always due to tuberculosis. This method, however, is of less importance than formerly, owing to the more definite, reliable, and earlier information given by the presence of bacilli. Calcareous fragments are sometimes expectorated along with the sputum.

According to Cabot the red corpuscles of the blood are normal in pulmonary tuberculosis and the hæmoglobin diminished. Stein and Erbmann show that leucocytosis is present under certain conditions. They think it is not connected with tubercular infection but is due to secondary infection. The physical signs of chronic pneumonic tuberculosis vary with the stage and extent of the disease.

Inspection.—The conformation of the chest may be indicative of disease though this form of tuberculosis occurs in chests of any shape or build. In the earlier stages inspection may be negative though a loss of expansion of the apices is noted very early in most cases. It is often best observed from behind the patient. As the disease progresses there is retraction of the supra and infraclavicular regions, flattening and widening of the second and third interspaces below the clavicle, marked loss of motion and drooping of the shoulders. If the upper lobes are emphysematous the scapulæ may be raised and the shoulders have an elevated appearance. The apex of the heart may be slightly raised, or displaced into the axillary region if much retraction of the left lung is present.

When a considerable cavity has formed in the apex the chest wall from the second to the fourth interspaces will be markedly flattened especially if there is induration and contraction of the tissues around the cavity.

Palpation is very important in the early stages. Slight increase in fremitus is obtained early. From the anatomical conformation and distribution of the right bronchus the right apex exhibits normally a better marked fremitus than the left apex. If the fremitus in the left apex is equal to that in the right it indicates disease; if it is greater, the indication is all the stronger. We must remember that a local area of moderate fibrosis combined with some degree of emphysema may, through better conduction facilities, give more marked fremitus than an area more advanced toward caseation but less easily vibrated. When a cavity has formed at the apex the fremitus is markedly increased. Palpation of the apices and lateral regions often gives a clear idea of the relative expansion of the two sides.

Percussion.—Slight increase in the pitch of the percussion note over the clavicles or in the supra or infraclavicular spaces is one of the earliest signs of the disease. It is often best appreciated when the breath is held after a full inspiration. Loss of resonance above the clavicle from retraction of the apex is often the earliest sign of a localized apical focus of disease. [There should normally be about a finger's breadth of resonance above the clavicle.] Dullness on firm percussion in the supraspinous fossæ may be present before it is apparent in front. If a cavity is formed the percussion note will be high-pitched, short, wooden, and tubular or tympanitic if the cavity is empty and the surrounding tissue is indurated. If the cavity is near the surface the cracked-pot sound may be obtained.

Auscultation.—Increased vocal resonance is an early sign of consolidation. Usually there is the same relative difference in the vocal resonance of the two apices as is found in the percussion pitch. The right apex often shows some increase in the vocal resonance without the presence of disease,—that is, above the accepted normal standard. Increased resonance is often best heard behind over the supraspinous fossa or toward

the point of the shoulder. Again, it may be best marked in the apex of the axillary space. Over a cavity the various sounds are cavernous or amphoric in character, and pectoriloquy may be obtained, often best heard with the whispered voice.

The earliest change in the respiratory murmur is often a feebleness or partial suppression of the inspiratory sound with prolongation of the expiration which is slightly higher in pitch than normal. Again, both sounds may be harsh and higher-pitched, the greatest elevation in the pitch being during expiration. Later, both sounds are much increased in pitch, the expiration is much prolonged and is tubular and blowing in character. Over areas of lobular consolidation bronchial breathing and bronchophony may be heard. In the tissue adjacent to the diseased areas wavy, interrupted or cogwheel breathing may be heard, especially if there is a tendency to fibrosis. It is often obtained in the lower and lateral regions of the chest. Over a cavity the typical breath sounds are cavernous in character,—a tubular, high-pitched inspiration followed by a prolonged, blowing expiration which is empty and tubular in quality and lower in pitch than the inspiration. The breath sounds may be markedly amphoric in quality.

Fine, crackling, subcrepitant, localized rales may be heard early, or more diffuse, moist rales may be present. As softening progresses the rales become more numerous, larger, softer and liquid in character. When rales are indistinct and doubtful in character they are heard more plainly with the respiration which follows the act of coughing. Gurgles and tinkling may be heard at times in cavities. Large cavities in the left apex may present the heart sounds very plainly, or even a transmitted systolic murmur. Systolic gurgles and clicks may be heard in a cavity if it is near the heart. A large cavity with thin walls and fluid contents may give a succussion sound (Walshe). Cardio-respiratory murmurs, and pleuro-pericardial friction sounds are frequently heard.

Areas of consolidation close to a large or dilated bronchus may give auscultatory sounds simulating those of a cavity in the lung tissue. Extensive retraction of the lower lobe with an emphysematous condition of the upper lobe may give

auscultatory signs simulating those of a cavity in the region of the interlobular septum. The lateral extension of these signs and the comparative condition of the two lobes will differentiate.

An absolute diagnosis may rest on the presence of bacilli in the sputum. While our knowledge of the relation of this organism to tuberculosis of the lungs greatly facilitates our diagnostic ability in doubtful cases, it is often the case that great attention is given to the demonstration of bacilli to the neglect of careful study of the physical signs and clinical history which is even more necessary to a thorough appreciation of the individual case.

Williams made early diagnosis of tuberculosis in 165 incipient cases with only two failures by means of the X-ray. He thinks the screen more trustworthy than photographs, and that the method is especially useful in scattered foci of disease with equivocal physical signs.

Many cases of insidious advent can be diagnosed by the physical signs before eruption into the bronchial tract exhibits bacilli in the sputum. Again, bacilli are sometimes absent from the sputum when there is well-marked local evidence of disease. Even in cases of moderately acute bronchial invasion bacilli may be temporarily absent while the apical localization makes it certain that the process is tubercular. We occasionally see local catarrhs in the apex presenting rales but no change in percussion pitch; which show no bacilli and which get well without leaving any evidence of permanent disease. However, any local change in the apex presenting raise in the pitch of the percussion note should be regarded as tubercular until it is proven otherwise.

In all cases of pulmonary tuberculosis bacilli will be found sooner or later if sought for, and in primary basic tuberculosis or in secondary tuberculous processes engrafted on chronic non-tuberculous basic lesions, the detection of bacilli may be the only way of deciding the nature of the process. In fibroid lesions—syphilitic or otherwise—accompanied with cavities the demonstration of the persistent absence of bacilli may be the sole means of proving the non-tubercular nature of the case. In cases with hæmorrhagic onset the early pres-

ence of bacilli may decide the nature of the case long before any local change can be detected.

Regarding the use of tuberculin for diagnosis, there is difference of opinion as to its practical value. Knopf thinks the older methods preferable to tuberculin and the X-rays. He calls attention to the undoubted fact that the possibility of a generalization of tuberculosis after the tuberculin test constitutes sufficient objection, as a rule, in personal cases in physicians. Otis' conclusions regarding tuberculin are: It gives earlier indication than the X-ray. The dose of Koch's original tuberculin is 10 milligrammes. This dose is harmless. Actual cases may fail to react with doses of 10-12 milligrammes. Syphilis may react. Non-tuberculous persons may give general reaction with a larger dose than the test dose. The reaction may be delayed for from six to twenty-four hours. Otis' rules are: Always use the same tuberculin and the same standard strength. Inject deeply into muscles with antiseptic precautions. Start a two, three, or four-hourly temperature chart twenty-four hours before injecting. Allow several days to elapse before a second test. In early cases depend upon the general reaction. In later cases if reaction fails look carefully for local signs.

Denison recommends a one per cent. solution of crude tuberculin in a seventy-five per cent. watery solution of carbolic acid (begin with a 1-2 mg. injection, rising to 25-30 mg.). Beck, of Koch's Institute, says the fear of disseminating tuberculosis by diagnostic injections is groundless. Head gives the reactions as 92 per cent. in pulmonary cases, 71 per cent. in cervical glandular, 88 in acute pleuritic, 100 in chronic pleuritic, 91 in arthritic tubercle, 100 in tubercular peritonitis, in suprarenal disease and in lupus.

COMPLICATIONS.—The complications which most often modify the course of pulmonary tuberculosis are pneumonia, bronchitis, pleurisy, pneumothorax, hæmoptysis, laryngeal tuberculosis, peritonitis, albuminuria and diarrhœa.

Pneumonia. Acute pneumonia is rare in pulmonary tuberculosis. It is a dangerous complication and while recovery may occur, the progress of the tuberculous lesion is hastened; markedly so if softening of the pneumonic area occurs.

Bronchitis. The danger from bronchitis usually lies in its liability to cause broncho-pneumonia or extensive emphysema. When bronchitis is extensive it may be the direct cause of death.

Pleurisy. The association of pleurisy with tuberculosis of the lung is frequent and its exact bearing on the history of the latter is not easy to define. Ocurring early in the history of the case it gives, according to Osler, "a stamp of chronicity to the case." Sero-fibrinous effusion may be absorbed. Purulent or hæmorrhagic effusions are very unfavorable. In general miliary tuberculosis, sero-fibrinous effusions may be an expression of the general disease.

In basic tuberculosis of the lungs the occurrence of pleurisy, if not purulent or hæmorrhagic, may modify the activity of the tuberculous process in the lungs.

Pneumothorax. The acute pneumonic form of tuberculosis is more apt to be complicated by pneumothorax than are the chronic forms. In rare instances of disease of the middle or lower lung the collapse of the lung incident to the occurrence of pneumothorax may limit the extension of the lesion of the lung.

When tuberculosis is limited to one lung the occurrence of pneumothorax may not be immediately dangerous, the air may be absorbed and partial re-expansion of the lung may take place. When the opposite lung is extensively diseased the occurrence of pneumothorax is dangerous and may be fatal in a few minutes. (In 39 cases of death after pneumothorax, the average duration of life was 27 days—Douglass-Powell.) In some cases life may be prolonged for years.

Hæmoptysis. In the early stages of tuberculosis the occurrence of hæmoptysis is not of immediate danger, in the later stages it is of grave import on account of the possibility of pulmonary aneurism. According to Williams, the percentage of deaths in a series of cases of hæmorrhage in the various stages was: in the stage of infiltration, 13.95; in the stage of softening, 24.61; and in the stage of excavation, 67.74.

Laryngeal Tuberculosis. This complication is generally unfavorable. It is usually of late occurrence. In cases where lesion in the larynx is primary the course of the disease may

be very rapid, while in other rare instances it may run a protracted course. The lactic acid treatment for laryngeal tuberculosis has modified the unfavorableness of the prognosis to some extent.

Peritonitis. Any form of peritonitis constitutes a very unfavorable complication of pulmonary tuberculosis. The most dangerous form is acute general peritonitis from intestinal ulceration with or without perforation. General tuberculosis of the peritoneum is rarely recovered from as a complication of pulmonary tuberculosis. Chronic localized tubercular peritonitis is less dangerous than the previous forms.

Albuminuria. The importance of albumen in the urine depends entirely on the cause as determined by careful analysis of the urine, and on the presence of changes in the vessels, heart, and other organs. According to Fowler, in cases of albuminuria associated with pulmonary tuberculosis, amyloid disease of the kidney was present in 6.1 per cent., parenchymatous nephritis, or interstitial nephritis in 6.7 per cent., and either miliary or caseous tubercles of the kidney in 6.5 per cent. of the cases.

Diarrhœa. Severe diarrhœa may be fatal in the late stages of pulmonary tuberculosis. It is commonly due to ulceration of the intestine, or to lardaceous disease. In either event the prognosis is very unfavorable.

TREATMENT.—In the treatment of pulmonary tuberculosis we include such measures of prophylaxis as are applicable to individuals who by birth or acquirement are predisposed to the disease, also such means of prevention of direct infection as apply with more or less direct force to every individual whether there be predisposition or not, and whose observance is demanded by public hygiene in order that the spread of pulmonary tuberculosis may be controlled.

As our inherent *vis medicatrix naturæ* yet remains the only specific we possess against pulmonary tuberculosis it follows that the treatment of the disease consists in aiding the forces of nature in every way possible to develop individual resistance against the disease.

The frequency with which quiescent tubercular lesions are found in the lungs of persons dying from other diseases

emphasizes the potency of the natural cure of tuberculosis. According to Heitler the Vienna post mortem records showed that in 4.7 per cent. of 19,292 deaths not directly due to tuberculosis the lungs contained old tuberculous lesions. Harris gives the percentage as 38.8 in 200 autopsies.

Prophylaxis. The prophylactic measures against tuberculosis should begin with the birth of the predisposed individual, if, indeed, they should not begin sooner. The results of sanitorium treatment in pregnant tuberculous females and their issue gives force to the statement of Knopf that the "State and municipal care of consumptives should begin with the child *in utero.*"

As far as the legal restriction of the marriage of tuberculous subjects is concerned there can be no question of the abstract benefit of requiring a clean bill of health in this particular despite disturbance of social ethics. The question of the marriage of persons with arrested tuberculosis of the lungs is not so simple. Females run much greater risks than males. It is better to give a straightforward opinion on the subject— which is usually disregarded—and place the responsibility where it belongs.

The child of a tuberculous mother should be suckled by a wet nurse or be fed artificially. Special attention must be given the dietary during the first few years of life. When fed on cow's milk it is best to boil the milk. Attention should be given to the nose and throat, to the bronchial affections of childhood, and to the general health after the infectious diseases of early life. The best prophylactic measure for these children is an out-door life. They should be literally turned out of doors to live in fresh air and sunshine. They should sleep in well ventilated rooms and have a daily cold sponge bath. As the period of puberty approaches we should be on the watch for conditions of anæmia especially in girls. Syrup of the iodide of iron, cod-liver oil, and arsenic are useful at this period. Later on, in early adult life, those who are predisposed to tuberculosis should be advised to remove to a suitable climate for a permanent residence.

Measures of prevention include the careful inspection of dairies, abattoirs and their products, by competent officers.

This is a matter of vast importance and one which state and municipal governments are coming to appreciate. Attention to the sanitary condition of public places and conveyances is also a matter where municipal regulations may be productive of much benefit.

The compulsory notification of pulmonary tuberculosis is a municipal question which has received considerable dis- cussion. As an abstract question of public health there is much to be said in its favor. On the other hand the parallel as to the necessity of notification between tuberculosis and the infectious diseases already on the notifiable list, is not readily apparent to the mind of the layman as the direct origin of the infection can usually not be demonstrated. Neither is the parallel a just one as far as the benefits accruing to the infected individual are concerned, nor will it be until there are state and municipal institutions for the treatment of indigent tuber- culous subjects. Sir Richard Thorne thinks compulsory noti- fication does more harm than good, that New York's experi- ence demonstrates it to be extremely partial, only effectual against tenement classes, and that the diminution of the death rate was already in progress. In the absence of the ability of departments of health to do anything more than furnish gratuitous information, it is doubtful if compulsory notifica- tion is advisable. The information can be equally well dis- tributed through the visiting and out-departments of medical institutions and such distribution should be required of them.

It should be impressed on the tuberculous subject that carelessness in his habits is dangerous to himself as well as to others. He should expectorate only into a sputum cup or cuspidor containing a five per cent. solution of carbolic acid, or into a small cloth which is to be immediately burned. He should under no circumstances swallow his sputum. The con- tents of the sputum cup should be burned two or three times daily and the cup boiled and disinfected. All eating utensils used by the patient should be washed separately and thor- oughly boiled, as should also all soiled linen worn by the pa- tient. Tuberculosis subjects should sleep alone.

Under proper precautions the dust from hospital wards containing tuberculous patients does not contain bacilli. The

same precautions applied to the home life of patients would render the danger of infecting others practically obsolete.

Our measures of treatment may be classed as general, special and symptomatic.

General Measures of Treatment. General methods consist in diet, tonic medication, antiseptic medication and open-air, sanitorium, and climatic treatment.

Diet. The nutrition of tuberculous patients is a most important matter. The dietetic rules must be adapted to the state of the patient's digestion. If the digestion is good a liberal, mixed diet is advisable. If the digestion is poor every effort must be made to improve it, and with this end in view a change of climate or a sea voyage may be undertaken with great benefit. When this is impossible the patient should be kept at rest, if there is fever, and fed every three or four hours with small quantities of milk, buttermilk, koumyss, eggs, meat extracts or powdered meat. As much time should be spent in the open air as possible. When the stomach is irritable the method of forced feeding recommended by Debove is useful. The stomach is washed out and a litre of milk containing an egg and 100 grammes of finely powdered meat is introduced through the tube thrice daily. If the patient is able to dispense with the tube the administration of a pint of milk containing an egg and a teaspoonful of somatose every four hours may be preferable.

Patients with active tubercular disease require a greater amount of nourishment than a healthy individual in order to compensate for the bodily waste. The results of forcing the amount of food as practiced at such sanitoria as that of Nordach in the Baden Black Forest are very encouraging when combined with the proper amount of fresh air and exercise.

A couple of teaspoonfuls of milkine in eight ounces of hot water taken at bed time or during the night often relieves the cough and promotes rest better than a sedative draught. The same potion in the morning promotes expectoration and counteracts the depression so common at this period of the day.

The routine administration of alcohol in tuberculosis is not advisable. Dry wines may be allowed with benefit in

some cases. In advanced stages a little champagne or whisky or rum taken in a hot drink may be of occasional benefit.

Tonic medication. The most reliable tonic remedies are cod-liver oil, strychnia, hypophosphites, iron, and arsenic. These remedies, as a class, are not adapted for the periods of high fever.

Cod-liver oil is of benefit, when easily assimilated, in improving nutrition. It is more acceptable to children than to adults and is better tolerated in winter than in summer. Its value in tuberculosis has been much over-estimated in the past. The oil alone generally disturbs the digestion before enough has been taken to be of any great benefit. It is best administered in capsules in combination with creosote, or some of the many "tasteless" preparations may be used. The following formula may be used for an emulsion of cod-liver oil:

R

Ol. morrhuæ,	℥vi.
Glycerini puri,	℥i.
Tinct. quillaiæ,	ʒiss.
Aq. lauro-cerasi,	℥i.

M. Ft. emulsio (to be shaken).

The dose of cod-liver oil should not be large enough to disturb the stomach. The so-called extracts of cod-liver oil appear to be as beneficial as the oil itself. After several years continuous use of them I am convinced that they stimulate digestion and improve the bronchial catarrh associated with tuberculosis to as great an extent as the oil itself when the latter is well borne. They are not irritating to the stomach and can be given to children without difficulty and can be given continuously at any stage of the disease with great benefit.

Strychnia is a valuable remedy in tuberculosis. Williams regards it as a specific for the retching of phthisical patients, and says it enables them to take cod-liver oil continuously without gastric derangement when given in doses of from 1/32 to 1/24 of a grain. Mays gives strychnia an importance next to rest and nutrition in phthisis. He gives it in doses as high as 1/7 of a grain. Pepper has reported excellent

results from the continuous use of strychnia. While Mays'
estimate is an exaggerated one, strychnia is a very useful
remedy. All other medicines seem to have a better influence
when the patient is under the effect of stimulating doses of
strychnia. A convenient method of administration is to be-
gin with six drops, thrice daily, of a solution containing two
grains to the ounce; increase one drop every third day until
the patient is taking as much as is well borne (usually about
15 to 18 drops). Insomnia is sometimes the first symptom of
overdosing.

The alkaline hypophosphites are valuable in pulmonary
tuberculosis. The combined hypophosphites have, after a long
period of trial, seemed to me to be of little use, indeed, in
many instances, seemed to be distinctly harmful. On the other
hand the hypophosphite of soda is of great benefit in many
cases. It should be given in doses not to exceed two grains
thrice daily, and is adapted to cases with little rise in temper-
ature. If a temperature of 100-101° F. is present the
hypophosphite of quinia may be used. Hypophosphites are
beneficial chiefly through their influence on nutrition. Daren-
berg says there is a great loss of hypophosphites from the body
in phthisis and thinks that they aid the formation of fibrous
tissue and the calcification of tuberculous formations. Hodg-
kinson concurs in this opinion.

Iron is a valuable tonic in tuberculosis. According to
MacKenzie the neutral preparations are best, and fever is re-
garded as a contraindication. Fowler recommends the syrup
of the iodide, or the phosphate, in cases with glandular en-
largement in young subjects. He thinks Blaud's pill is useful
if cough is not severe, but thinks that iron increases the cough
and the tendency to hæmorrhage. I have found the so-called
"tasteless" tincture or the citro-muriate of iron, combined
with the compound tincture of cinchona, of value in tubercu-
lous cases.

Arsenic is of use in some cases. Williams recommends
it in the early periods and in cases combined with asthmatic
conditions. Osler advises the use of Fowler's solution in all
varieties of tuberculosis. Fowler and Burney Yeo have not
seen much benefit from the use of arsenic. Arsenic is of par-

ticular value in anæmic cases and the iodide of arsenic is some-
times of special value. On the whole arsenic does not seem
to be as efficient in combating the malnutrition of tuberculosis
as it is in the malnutrition of other conditions and Jaccoud's
statement that "It would be difficult to find an agent more
capable of combating the malnutrition always existent in the
disease" seems to be an overestimate of the value of the drug.

Iodoform is, according to Ransome, one of the best drugs
for aiding nutrition and relieving cough. Europhen may be
used as a substitute for iodoform. Flick uses an inunction
composed of

Europhen,	ʒi.
Oil of rose,	gtt. i.
Oil of anise,	ʒi.
Olive oil,	ʒiiss.

A tablespoonful of this is rubbed into the arm-pits and
thighs at night. The odor may be removed in the morning
by sponging with bay rum.

Antiseptic Medication. It is generally admitted at pres-
ent that it is impossible to directly inhibit the tubercle bacil-
lus through the internal administration of anti-bacillary rem-
edies, and that the good effect of these remedies when given
internally is through their effect on nutrition by stimulating
digestion and assimilation. Foxwell considers iodoform the
most satisfactory of antiseptic drugs. Knopf's formulæ for
iodoform are:

℞

Iodoformi,	1 part.
Ætheris,	10 parts.

As an inhalation. Twenty minims to be used on a respir-
ator at each inhalation.

℞

Iodoformi,	gr. ss.
Codeinæ sulph.,	gr. 1/3.
Et. cascara sag.,	gr. ss.

M. Ft. pill.

℞

Iodoformi,	gr. xxx.
M. Tannin,	gr. lx.

To divide into twenty cachets; from two to four cachets daily.

Creosote is the principal remedy of this class. Known since 1830, and its use in tuberculosis described by Bouchard and Timbert in 1877, it was not much in use until Summerbrodt in 1887 gave the results of the treatment of 5,000 cases with creosote. Summerbrodt believed in its specific properties and ardently advocated its use in immense doses. It is generally conceded that moderate doses give better results than are obtained by attempting to saturate the system with the drug. The best results seem to be obtained from doses not to exceed from five to ten minims. Pure beechwood creosote is best. It may be given in capsules, or the creosote "perles" may be used, or it may be given in cod-liver oil, malt, or wine with vegetable bitters. The most convenient method is to drop it into empty capsules and follow its administration with a glass of milkine. Carbonate of creosote (creosotal) is a more eligible preparation in that it has not the disagreeable odor of creosote and is not so irritating to the stomach. It is a liquid, has little taste, is said to contain 92 per cent. of creosote, and in the intestinal canal it splits up into creosote and carbonic acid. It may be given beaten up with the yolk of an egg, or may be given in capsules. The dose is, at first, five drops after meals. Leyden's method is to increase three drops daily up to twenty-five drops; after two or three weeks it is reduced gradually to ten drops, and subsequently the dose is again increased.

Guaiacol, a derivative of creosote, is much used as a substitute for the latter. Aside from its odor it is a more eligible preparation than creosote. It agrees better with the stomach, and it has seemed to me, after several years' continuous use of guaiacol, that the results were better than with any other preparation of this class except with the pure creosote in such patients as tolerated the creosote unusually well. Guaiacol may be administered in the same way as creosote and in about the same dose. From five to ten drops appear to give about the best results. The following is a useful formula:

Guaiacol,   ℥ii.
Alcohol,   ℥v.
Wine of cod-liver oil,   ℥xii.
     Dose, a tablespoonful.

The following formula is an eligible one for preparing wine of cod-liver oil:

Gaduol,	64 grains.
Guaiacol,	128 minims.
Alcohol,	1 fl. oz.
Fuller's earth,	4 drams.
Peptonized manganese,	2 drams.
Water,	2 fl. oz.
Sugar,	2 oz.
Port wine,	q. s. to 1 pint.

Mix the gaduol, guaiacol and alcohol and triturate thoroughly with the Fuller's earth. Dissolve the pep. manganese in the water, add the wine and sugar and mix thoroughly with the gaduol and guaiacol mixture. Let this mixture stand for a day, shaking occasionally. Filter and pass enough wine through the filter to preserve the volume.

Guaiacol may be given hypodermically, using 1 to 7 minims of sterilized guaiacol and injecting deeply. It may also be used by inunction, giving, once in two or three days, 10 to 60 minims either undiluted or with olive oil, glycerine, or tincture of iodine. These are, however, objectionable methods with no obvious advantages.

Carbonate of guaiacol may be given in cachets, or in capsules in doses of from five to fifteen grains thrice daily. It is well borne by the patient but does not appear to be as efficacious as liquid guaiacol. Benzosol is an antiseptic of value but in my experience has not been of special value in tuberculosis. The dose is from five to ten grains. Guaiacolate of piperidine is recommended in doses of from five to twenty-five grains. According to Chaplin and Tunnicliffe it is safe, causes no unpleasant effects, is exceedingly well borne, and patients under its influence improve in appetite and strength. Iodoguaiacol camphorate, eosote (valerianic acid ester of creosote) and various other combinations of creosote are recommended but do not appear to have special therapeutical advantages or power over those already mentioned.

The use of antiseptic inhalations and sprays is of advantage in pulmonary tuberculosis in lessening the amount

of expectoration, modifying the cough, and preventing infection of the larynx. Burney Yeo recommends creosote, carbolic acid and chloroform for the latter purpose. Fox thinks antiseptic inhalations of value. Eucalyptol, iodine in ether, menthol, aristol, oleum pini sylvestris, camphor, etc., may be used. Douglass recommends the use of eucalyptus oil made from the leaves of the eucalyptus globulus and administered by saturating the air of the patient's room by means of suspended cloths dipped in the oil, and by the inunction of three or four drams of lanoline ointment containing three drams of oil to the ounce.

Inhalations of the oil of cinnamon is highly recommended by Lucas-Championniere, and by Hilton Thompson. The results are said to be diminution of cough, expectoration, temperature, number of bacilli, and increase in weight.

Intra-tracheal injections of eucalyptol, menthol and other substances have been practiced with alleged good results. While this method is of benefit in bronchiectatic conditions in phthisical cases, it does not appear to be of any special benefit to the disease of the lung. Injections of a dram of a solution composed of guaiacol, 2 parts; menthol, 10 parts; olive oil, 88 parts, may be used.

The direct injection into the lung tissue of iodine, carbolic acid, creosote (3 per cent. sol. in sterilized olive oil), iodoform and other substances has been practiced and is strongly recommended by Pepper. When carefully administered such injections are, as a rule, not dangerous though serious results may follow them. Treatment by compressed air either pure or saturated with creosote has been attended with benefit in some cases. It has, however, not yet been demonstrated that the introduction of antiseptic substances into the lungs by any of these methods is attended with any permanent improvement of the diseased area of lung tissue, and while it is possible to introduce, by inhalation, oil solutions directly into the smaller bronchi, and, possibly, the alveoli also, the benefit, in phthisis, is probably confined to those tissues which are in direct contiguity with the patent air passages.

When we carefully review the equivocal position of the

old and tried medicaments for pulmonary tuberculosis, as well as the thousand-and-one modern "cures," we are obliged to admit that we are not yet in position to dispute Laennec's statement that "although the cure of tuberculosis is possible for nature, it is not so for medicine."

Fresh Air Treatment. The benefit to the tuberculous subject of the systematic indulgence in fresh, pure air is universally conceded by all observers. This recognition of the power of fresh air is admitted in no equivocal terms. Wilks says "The only remedy known for consumption is air, air, fresh air." Ransome says "Fresh air night and day before everything else." Trudeau's experiments on confined and unconfined rabbits illustrate the value of the open-air treatment of tuberculosis.

The fresh air treatment may be carried on with the patient at home or at a suitable sanitorium, without a change in climate. At home the fresh air treatment is more or less difficult to manage, especially in the cities. A partially successful attempt, however, is better than to house a patient in a stuffy, ill-ventilated room, dope him with medicine and blame nature for the result. The patient should have the sunniest, brightest room obtainable. The windows should be kept open during the day, and partially so at night. If there is active disease with fever the patient should be kept in bed, or if there is a porch or yard available he may be wrapped up and placed in an invalid's chair and allowed to spend four or five hours daily in the open air. Neither cough, fever, night sweats, or hæmorrhage contraindicate fresh air. Ransome says that nothing relieves the fever so much as fresh air. Moderately low temperature is not important, but wetness and high winds should be guarded against. If the patient is well enough to be about he should spend six to eight hours daily in the open air and take a daily amount of exercise which must be specified by his physician.

The sanitorium treatment of tuberculosis has risen in importance because of the results obtained at such institutions as those of Nordach, Falkenstein, Göbersdorf, Davos, etc., in Europe, and at the Sharon sanitarium near Boston, the Adirondack sanitarium, the Asheville sanitorium, the Aiken

sanitarium, etc., in this country, and various institutions in Canada. To obtain good results it is not necessary that the sanitaria be situated in an ideal climate. Burton-Fanning says that good results are obtained where climatic conditions are only moderately favorable, though the conditions most conducive to good results are pure, cool, bracing air, abundance of sunshine, dryness of soil, and elevation above the sea. At the Victoria hospital for consumptives, near Edinburgh, where the climate is by no means ideal, and the patients not of the most favorable class, with an average residence of less than three months, the results are said by Philip to be very encouraging. The plan of treatment in these sanitoria is to keep the patient in the open air during the day as much as possible without regard to the weather except in special cases. Very liberal diet is given. The windows of the patient's room are wide open all day, and sufficiently so at night to insure good ventilation. The average temperature of the rooms is kept at from 60-65° F. Exercise, bathing, medication and all auxiliary means are employed to hasten recovery. Daily observation is given each patient. These institutions, conducted under the supervision of competent and reliable men, in whom the profession may repose confidence, furnish better opportunity for recovery to a patient than he will find by adopting climatic treatment in his own way, and without intelligent direction.

There is great need in this country for sanitoria near our large cities where patients with moderate incomes may be treated at a small expense. It is to be hoped that the state and municipal governments will soon realize that there is not only humanity, but also economy, in the establishment of public institutions on the sanitorium plan for the treatment of their indigent tuberculous residents.

In the climatic treatment of tuberculosis the open air feature must not be neglected. Patients sometimes have the idea that if they make a suitable change in climate they must perforce get well without any effort on their part. The value of any climate is in direct ratio to the number of days throughout the year which the patient can be in the open air and sunshine. The average temperature is of secondary importance.

The majority of the patients with good family history and in the early stage of the disease will get well in any climate if they live an outdoor life.

Proper accommodations and diet are not always obtainable in desirable climates and this must be considered when patients are not able to endure an out-door life. Those who can live out doors can usually get along with such food as can be obtained. A camping trip across our western or southwestern states is one of the best methods of utilizing the fresh air and climatic methods of treatment.

The earlier the climatic change is made the better the chances for recovery. It is very difficult to persuade patients to break up business and social associations early enough to give them the best results from climatic treatment. Later, they are not only willing, but insist, against advice, on a change of climate. In case of improvement it is difficult to keep patients in a proper climate for a sufficient length of time. Properly conducted sanitoria in our own localities would obviate some of these difficulties by affording a place where patients could be kept under proper conditions for a portion of the year.

In deciding the advisability of a change of climate we must be governed entirely by the patient's condition, and not by the anxiety of himself and friends. Patients with high fever, night sweats, loss of flesh, extensive areas of lung involvement, or cavity formation, should not be allowed to go away from home.

The various climates may be divided into those of high altitudes, the dry, warm climates, and the moist, warm climates.

High altitudes possess, according to Williams, diminished barometric pressure—rare atmosphere, and diathermacy or increased facility of transmission of the sun's rays which causes an increase in the difference between sun and shade temperature of $1°$ F. for every rise of 235 feet. The general result in selected cases of chronic tuberculosis being excellent: improvement in 83 per cent., great improvement in 74 per cent., disappearance of all symptoms in 41 per cent.; local conditions show 75.5 per cent. improved, 5.5 per cent. stationary,

and 19.18 per cent. worse. Both sexes do well. The most favorable age is from 20 to 30, and those with hereditary disease are especially benefited.

Cases of incipient disease, with hæmorrhagic tendency, are often much benefited by high altitudes, though such persons should not be suddenly changed from a low to a high altitude. Patients with a temperature above 101° F. should not be sent into high altitudes, and the latter is contraindicated in patients with extensive lung involvement, cavities, aneurism, laryngeal involvements, catarrhal tuberculosis, all varieties of acute disease, weak heart, emphysema, and chronic varieties of tuberculosis when much lung tissue is involved or there is extensive degree of fibrosis of the lung, particularly where the heart is weak.

A residence of from eight to twelve months at least is necessary in order to obtain benefit from high altitude. Of the higher altitudes in this country the Colorado, New Mexico and Arizona resorts are the best. Asheville and the Adirondacks are the best of the more moderate elevations.

Such moist, warm climates as Florida (Gulf coast), the Bermudas, the Madeira Islands, the Island of Corsica, and Falmouth in Great Britain are the best. They are adapted to cases with limited lung space, emphysematous and bronchial conditions, and weak hearts.

Dry, warm climates, like the lower portion of the Rio Grande Valley, southwestern Arizona, southern California, various points in Georgia, the Carolinas and eastern Tennessee in this country; the Riviera, Egypt and Algiers are the most noted points. According to Foster it is doubtful if the Mediterranean offers the best climate for the majority of cases. Hillier praises South Africa, and Sandwith enumerates the advantages of the Egyptian deserts.

Ocean voyages, while beneficial in some cases, are contraindicated, according to Parkes Weber, in advanced cases, cases with fever, in laryngeal and in intestinal tuberculosis, and in cases associated with advanced arterial sclerosis or cardiac disease.

Special Treatment. The treatment of tuberculosis with special preparations like the new (T. R.) tuberculin, Mar-

agliano's anti-tuberculosis serum, Hirschfelder's oxytoxins, etc., is yet in an experimental stage, and the one thing definite in regard to it is that the literature of the past five years on this subject will make curious reading half a century hence. Trudeau, whose experimental work with various serums at the Saranac laboratory gives his opinion weight, thinks that the outlook for an efficient tuberculosis antitoxin is not entirely hopeless.

Symptomatic Treatment. The symptoms which most often demand attention are the fever, cough, sweating, diarrhœa, hæmorrhage, and gastric disturbances.

Fever is almost constantly present in pulmonary tuberculosis and is, to some extent, a measure of the intensity of the disease. Its reduction is a most difficult therapeutic measure, indeed, so little is accomplished toward the well-being of the patient by the reduction of the fever by drugs that it is only when the fever is unusually high that it should be attempted. The depression resulting from the use of the coal tar products overbalances the slight benefit derived from them. The combination of two and a half grains each of phenacetine and quinine is often of use given every three or four hours or every two hours for three doses just before the rise in temperature in a septic type of fever. Quinine alone must be given in large doses which are not always well borne. When the fever is above 101° F. the patient should be kept at rest, with as much fresh air as possible. Ransome says that nothing, in his experience, relieves the fever so much as fresh air and sponging with cold water and vinegar. Fever is no contraindication to fresh air and as much of the latter should be had as is possible without overexertion. Sponging with tepid cologne water is grateful and useful.

Cough is a troublesome symptom and one which patients are usually anxious about. It is well with the cough, or with the fever, to trust to general measures as much as possible for relief. If cough is troublesome at bed time, or paroxysmal in the morning, a cup of hot milkine will often afford much relief. Nauseous cough mixtures are to be avoided, and when cough medicines are prescribed they should be as simple as possible. In nervous, irritable people Hoffman's anodyne is

often very useful. A mixture containing to each dose ⅛ gr. of morphia or ¼ gr. of codeia, 10 drops of dilute hydrobromic acid, and syrup of tolu q. s., will be taken longer, with more relief and less complaint, than most other cough mixtures. If the cough is hacking and very persistent we may use ¼ gr. of codeia, 1 gtt. fl. ext. of cannabis indica, 3 grs. of muriate of ammonia, and syrup of tolu q. s. In cases with considerable bronchitis and scanty expectoration we may use muriate of ammonia, gr. 5; codeia, gr. 1/6; tincture of pulsatilla, gtt. 6 to 8; liq. am. acetat., gtt. 20; elix. simp., q. s. In laryngeal cough we may use inhalations, sprays and sedative troches. Oil solutions of the various antiseptic drugs are of great use in relieving cough and expectoration when given by inhalation. Warm applications or counter-irritation to the chest may be useful. Lozenges of acacia and licorice are useful in laryngeal cough. Spraying with a 2 to 3 per cent. solution of menthol or aristol; inhalations of 20 drops of a saturated solution of menthol, or a like amount of equal parts of creosote or guaiacol with spirit of chloroform when the membrane is dry and irritated may be of use. With laryngeal catarrh we may use a teaspoonful of compound tincture of benzoin in a pint of water near the boiling point for inhalation. With profuse secretion from the mucous membrane we may find 3 to 5 drops of tincture of belladonna and half a teaspoonful of syrup of senega every four hours of value.

Night sweats are a troublesome through erratic symptom in tuberculosis. When regular and severe they weaken the patient and should be stopped if possible. A cool sponge bath in the morning followed by thorough friction of the body will often lessen the sweating. Atropine is the most useful remedy. It should be given at bed time in 1/60 to 1/50 grain doses. Camphoric acid is, next to atropine, the best remedy for sweating. It is best given in three doses of five or ten grains each (in capsule) at 5, 7 and 9 o'clock in the evening. Agaricin (1/12-1/10 gr.) may be given six hours before the sweating takes place, or, as I have found useful, 1/15 of a grain may be given at 2 and 7 P. M., followed by thirty drops of fluid extract of pinus canadensis at bed time. Picrotoxin (1/60 gr.) may be given at bed time. Zinc oxide (5 gr. in

pill) is sometimes useful. If profuse sweating occurs whenever the patient sleeps it is an indication for stimulants and nourishing drinks.

Diarrhœa may be troublesome when there is acute or chronic ulceration of the intestines. In the acute form ten grain doses of bismuth (sub-carbonate or sub-gallate) with five drops of deodorized tincture of opium and a like amount of spirits of camphor every three or four hours is useful. Dover's powder with bismuth is of service. Starch enemata, plain or with opium, may be used. The acetate of lead and opium pill is useful. If the discharge is watery and profuse ten drops of dilute nitric acid with five drops each of tincture of opium and tincture of camphor given in simple elixir will be of use. In the diarrhœa of chronic ulceration the diet is very important. Such drugs as acetate of lead (2 to 3 gr.), sulphate of copper ($\frac{1}{4}$ to $\frac{1}{2}$ gr.) combined with opium in pill form, are of use. Bismuth (gr. 20), and co. ipecac pulv. (gr. x) may be given in milk every 4 to 6 hours. Tincture of catechu, gtt. 20; tinct. opii deodor., m. 5; mist. creatæ, ℥ iv, may be tried. Fluid extract of coto bark in five minim doses may be useful. When the lower intestine is affected lead and opium pill combined with starch enemata (tr. opii, gtt. xx to ℥ ss; mucil. amyli ℥ ii) is of great service.

Hæmorrhage is to be treated by rest and opium (morphine hypodermically). If the vascular tension is high lower it with aconite and sodium bromide. No stimulants should be given. Tincture of hamamelis in 20 to 60 minim doses is recommended. In profuse hæmorrhage, with low temperature, we may use ergotin hypodermically. Temporary ligation of the four extremities is efficacious in profuse hæmorrhage, and from 1/50 to 1/30 of a grain of atropia may be given hypodermically. Astringents, as a rule, do no good in the pulmonary hæmorrhage of tuberculosis.

Gastric disturbances are to be avoided, to a large extent, by the general treatment and attention to diet. Acidity and flatulence may be relieved by bismuth and bromide of sodium or strontium. For loss of appetite without digestive disturbance Fowler recommends bicarbonate of soda, gr. xv.; dilute hydrocyanic acid, m. iii; comp. inf. of gentian ℥i.

Gastric crises may occur which must be tided over by restricting the diet to liquid food. Vomiting may occur through the irritation produced by the severe coughing or through some sore spot or sensitive area in the throat, attention to which will relieve the vomiting. The nausea so frequently present may be controlled by small and frequent doses of lime water containing one drop of carbolic acid to the ounce, or by small doses of calomel and soda.

Pain from pleurisy may be controlled by strapping the chest, painting the surface with tincture of iodine, applying belladonna plasters, small blisters, or hot applications.

The experimental work of Biondi, Schmidt, Gluck and others show that a portion or the whole of a lung may be removed without fatal results. Lowson and Tuffier have successfully removed a tuberculous portion of the human lung with good results. While these cases demonstrate the possibilities of lung surgery, the application of such methods must be extremely limited in the treatment of tuberculosis of the lungs. The surgical drainage of tubercular cavities of the lung has generally been regarded with disfavor. It is not beyond argument, however, that this may be the most advantageous way of treating single cavities properly situated. Should the future discover a reliable bacillicide for the after treatment of drained tubercular cavities the question of operation would assume a different aspect.

CHAPTER XV.

ACTINOMYCOSIS OF THE LUNGS.

Bollinger described actinomycosis in 1877, and in 1878 Israel described it as affecting the lungs, though the disease had previously been observed in man by von Langenbeck in 1845, and by Lebert in 1857. The disease is said to be somewhat more common in Germany and Russia than in this country, and to be rare in England. It is much more common in men than in women. The total number of cases of actinomycosis now on record is comparatively small.

Pulmonary actinomycosis is an infectious disease of chronic course caused by the actinomyces, or ray-fungus (*streptothrix actinomyces*).

ÆTIOLOGY.—The fungus which causes the disease gains entrance to the body through the respiratory or alimentary tracts. In the latter case the symptoms may be chiefly thoracic, as, when the disease affects the liver it may extend to the pleura and lungs and open through the ribs. The organism has not been recognized outside of the body. There is no direct proof of infection through the flesh or milk of diseased animals. It is most likely that the infection occurs through the medium of the food.

It is not positive as to just which group of bacteria the organism belongs, though it is credited to the streptothrix group. It is markedly polymorphous and occurs as small spherical or irreguarly-shaped grains or masses of a sulphur-yellow color, and from one-half to two millimetres in diameter. These bodies present an internal granular mass and an outer zone of radiating mycelia which may present bulbous terminations. According to Boström the clubbing is due to hyaline degeneration of the filaments. These small masses which are characteristic of the disease are found in the granulations of the diseased area, or in pus from such area. In the early stages of their formation they may be grayish in color.

MORBID ANATOMY.—The lesions of pulmonary actinomycosis are unilateral in the majority of instances. According to Hodenpyl there are three groups of lesions: lesions of chronic bronchitis; miliary lesions resembling miliary tuberculosis, the nodules are composed of groups of fungi surrounded by granulation tissue; destructive lesions of the lungs characterized by broncho-pneumonia, interstitial fibrosis, and abscess of the lungs.

When the chest wall becomes involved there is enlargement of the affected side, effacement of the intercostal spaces, the soft tissues become discolored and œdematous, the skin becomes red in spots, and abscesses form and discharge a small amount of pus. Fungoid masses of red and yellow granulation tissue form around the openings.

Actinomycosis of other organs and tissues is frequently associated with the pulmonary form of the disease. Necrosis and erosion of the ribs, vertebræ or sternum; subcutaneous abscesses, and metastases to other portions of the body occur.

CLINICAL HISTORY.—The clinical history of pulmonary actinomycosis may be similar to that of pulmonary tuberculosis, tumors of the lungs, or chronic pleurisy. The fact that actinomycotic and tuberculous processes may be associated in the lungs tends towards ambiguity of the clinical history.

The onset of the disease is gradual and is accompanied by an insidious cough, with, perhaps, slight expectoration. There is general weakness with loss of weight. Pleurisy, with or without effusion, may be a feature of the early stages. The patient becomes pale and anæmic and loses flesh; cough becomes more troublesome, and the sputum becomes muco-purulent, more abundant, and may contain the yellow granules which are characteristic of the disease. The temperature is more or less elevated and gradually assumes a hectic type.

When the chest wall becomes involved there is a change in the contour of the affected side. Scoliosis may develop. The soft tissue becomes swollen and inflamed, abscesses form and discharge pus and the sinuses are surrounded by granulation tissue. The clinical history is now much like that of caries of the ribs or vertebræ. The symptoms may resemble those of emphysema, or may simulate those of typhoid fever.

The course of the disease is progressive. The average duration is about one year. Recovery is very rare. Death is preceded by general septic conditions.

SYMPTOMS AND DIAGNOSIS.—Cough, fever, emaciation; with muco-purulent or perhaps fetid expectoration are the principal symptoms. Cough and expectoration are important symptoms as in some cases the diagnosis may be reached through the presence of the actinomyces in the sputum. Bizzozero cautions against mistaking degenerated epithelial cells in the sputum for the organism of the disease. The fever is irregular in type and eventually becomes hectic. The physical signs are usually indefinite. The lower portion of the lungs is the part usually affected, and the disease is generally confined to one side of the chest. More or less localized dullness will be present and there will be a corresponding loss of respiratory sounds. Signs characteristic of pleural effusion may be present. There may be signs of a cavity in the lung. As the disease progresses and the chest walls become affected there will be much restriction of motion and the voice and respiratory sounds will be weak and indefinite.

In the early stages it may be impossible to differentiate the disease from tuberculosis, or from chronic pleurisy. The presence of actinomyces in the sputum, or in the discharge from the chest wall may be the only means of making a positive diagnosis. If the disease has extended from the liver the latter will be enlarged. Extension from the upper respiratory tract, or from the mouth may render the diagnosis less difficult than usual.

Godlee says that if, on opening an abscess connected with the ribs, pleural cavity, or spine, the amount of pus is small and hæmorrhage unusually free, and especially if subsequent insidious burrowing of pus beneath the skin occurs, we should be suspicious of actinomycosis.

TREATMENT.—The medicinal treatment of actinomycosis of the lungs is symptomatic only. Large doses of iodide of potash (20 to 40 grains t. i. d.) are recommended but it is doubtful if it does any real good. Injections of iodide of potash have also been recommended.

Surgical measures are the only means that may avail

against the inevitably fatal results, and on this account much greater risk may be assumed than is justifiable in other pulmonary diseases. We must bear in mind the great danger of hæmorrhage during operation, and also the possibility that the disease has extended to places and tissues where surgical intervention cannot follow.

MYCOSIS PULMONUM.

Pulmonary mycosis (pneumonomycosis) is a disease of the lungs caused by the presence of fungi. The disease may be primary or secondary (fungus growth in the cavities of phthisical lungs).

Virchow first recognized the disease in 1854. The principal form of the disease is that due to the *aspergillus fumigatus*. Other forms may be due to sarcinæ (p. sarcinica), to an oidium, which is said by Gilchrist and Stokes to be allied to the yeast fungus, to an organism called by Flexner the streptothrix pseudo-tuberculosa, and, according to some observers, to some of the mucosinæ or moulds. Rixford and Gilchrist have reported cases of protozoic infection of the lungs.

These various infections of the lungs are followed by changes in the lung tissue which are very similar to those occurring with tuberculosis. The symptoms and clinical history are those of chronic lung disease and may closely simulate those of tuberculosis, chronic bronchitis, asthma, hæmoptysis, etc. The diagnosis depends on the demonstration of the nature of the infection.

Rénon and Dieulafoy hold that the aspergillus fumigatus may be a pathogenic organism, though in some cases it is secondary. Virchow and others maintain that it is always saprophytic, and the lesions are secondary to other processes.

Secondary aspergillo-mycosis may occur in connection with pulmonary tuberculosis, chronic bronchitis, bronchiectasis, hæmorrhagic infarction of the lungs; malignant growths in the lungs, and to valvular lesions of the heart.

HYDATIDS OF THE LUNGS.

Hydatids of the lungs (echinococcus of the lungs) is due to the presence in the lungs of the larvæ of the tænia echinococcus. The disease is most common in Iceland, Australia, and some European countries where there is close association between men and dogs. It is rare on this continent, though Ferguson states that a number of cases have been observed in Winnipeg since the Icelandic immigration in 1844.

ÆTIOLOGY.—The cestode of the tænia echinococcus is four or five mm. long and consists of three or four segments. The terminal segment is mature and may contain 5,000 eggs. The head is small and has four sucking disks surrounded by a double row of hooklets. The ova are accidentally ingested by man, chiefly through the medium of the drinking water.

The ovum having reached the stomach is relieved of its envelope by digestion and the embryo makes its way through the coats of the stomach or intestines and may reach the lungs, after passing through the capillaries of the portal or hepatic veins, by way of the pulmonary artery, or, it may enter a systemic vein after leaving the stomach, and thence gain the pulmonary artery. According to Bird, hydatid disease of the lung may occur through inhaling dust containing the ova of the echinococcus, in which case they might enter the bronchial arteries. It is not definitely known, however, how much of their movement is to be attributed to migration and how much to vascular transportation.

Echinococci are more frequent in the lung than in the pleura, but the liver is the favorite seat of the affection. In this country about seven per cent. of the cases have occurred in the lungs or pleuræ. In 1,862 cases collected by several observers the lungs and pleuræ were affected in 153 cases, Thomas's collection of 809 cases show the lungs to have been affected in 134 cases, and the pleuræ in 2 cases.

MORBID ANATOMY.—When the embryo has reached its destination its hooklets disappear and it is gradually changed into a small cyst containing a clear fluid. The wall of the

cyst is composed of an external thick, transparent, elastic, laminated layer (ectocyst); and an internal granular, paren-chymatous layer (endocyst). This cyst determines more or less formative inflammation in the surrounding tissue, and becomes enveloped in a fibrous capsule. This capsule is rarely as dense as it is in hydatids of the liver, and frequently it is slight or absent altogether.

The method of development of the cysts is the same as in other tissues and need not be considered here. The fluid contained in the cysts is clear and limpid and has a specific gravity of 1.005 to 1.015. It contains no albumen, or at most only a trace. There may be traces of sugar. Usually the cysts contain scolices and the characteristic hooklets.

As the cysts grow they compress the lung tissue; cause inflammation of the surrounding tissue, gangrene, and the for-mation of cavities which may be connected with the bronchi. The bronchi may be patent directly to the surface of the cyst, but as long as the latter is tense with fluid the bronchi will not open directly into the cyst. If the cyst collapses the bronchi open and the air enters the cyst cavity.

Rupture into a bronchus may occur with discharge of the cyst contents, or even of the cyst itself, in which case the cavity may become obliterated. This result is rare, however, for, if the cyst is surrounded by a fibrous wall the cavity con-tinues to discharge pus. Rupture into the pleura is followed by pneumothorax, and pleurisy with serous or purulent effu-sion. Rupture into the pericardium is usually rapidly fatal.

The lung tissue surrounding a cyst may become pneu-monic, gangrenous (if the cyst is dead), or fibrotic. These changes may lead to the death of the cyst which may in time be followed by suppuration and decomposition of its contents. It thus becomes an abscess which may discharge in any direc-tion. If the cyst dies, its contents become turbid and albumin-ous. The adult cyst may become glutinous, opaque and may break up into fragments. If there are daughter cysts, they will later pursue the same course. In rare instances the cyst contents may undergo fatty degeneration and the cyst dries up.

Hydatid cysts of the lung are usually single. The mul-tilocular variety is very rare. Both lungs may be involved,

and there may be more than one cyst in a lung. The right lung is more often affected than the left; and the lower lobes more frequently than the upper. Very few cases of hydatids of the lungs recover.

CLINICAL HISTORY.—Small cysts in the lung may give very indefinite symptoms, or none at all. There may be a history resembling that of tuberculosis. Usually there is a dry, hacking, paroxysmal cough with some mucoid expectoration. Slight hæmoptysis is quite common in the early stages; later on it may be profuse. Fever is generally absent unless inflammation is present in the lung. Moderate dyspnœa may be present, but the dyspnœa is not troublesome unless pressure is exerted on large vessels. Pain is slight or absent unless the pleura is involved. There may be a sense of weight or constriction about the chest. Loss of flesh and weight is common. Rupture of a cyst into a bronchus is attended with severe, sudden paid in the chest, violent cough, marked dyspnœa, the expectoration of watery fluid which contains the characteristic hooklets, and possibly by severe hæmorrhage. Small pieces of hydatid membrane may be present in the expectorated fluid. If the bronchus opening into the cyst is small the escape of the contents of the cavity may be gradual and accompanied by less severe symptoms. If daughter cysts are present in the expectorated fluid the bronchus entering the cavity is probably a large one. Hæmorrhage may occur at intervals after the rupture. Death may occur at the time of rupture of the cyst. If communication be established with the pleural cavity the signs of pneumothorax and pleurisy will follow.

After rupture of the cyst the violent, severe and paroxysmal coughing spells may continue, the expectorated matter containing fragments of membrane and daughter cysts. The expectoration may become purulent and fetid, or it may be dark-colored and resemble pus from an hepatic abscess, while the clinical history may be similar to that of gangrene of the lungs. Hectic fever, loss of flesh, and clubbing of the finger ends may occur. In some cases the symptoms are much less severe, and aside from the dyspnœa which may attend the expectoration of cysts or fragments of cyst wall the history may not be distinctive.

SYMPTOMS AND DIAGNOSIS.—A small, deeply-seated cyst may give no symptoms. If a large cyst exists there may be diminished chest expansion and respiratory murmur. If the cyst is near the surface there will be loss of expansion, diminished voice and respiratory sounds, slightly tympanitic or a flat percussion note, and diminished or absent vocal fremitus. The cardiac impulse may be displaced and the chest wall may show local prominence and effacement of the intercostal spaces, or the cyst may project through a space and form a round tumor on the chest wall. According to Fowler, the most characteristic sign is a round area of dullness on percussion showing absolute dullness in the center with gradually increasing resonance toward the margin; the breath sounds and vocal resonance being absent over this area. If the cyst is near the chest wall the so-called hydatid thrill may be felt.

Sub-crepitant rales from œdema and congestion of lung tissue may be heard. Signs of pressure on the vessels, pneumogastric nerves, or on the œsophagus may be present. The differentiation from tuberculosis rests on the location of the lesion, its failure to involve other portions of the lungs, by the absence of bacilli and by the greater effect of tuberculosis on the general health.

Signs of a cavity may be obtained soon after rupture and before contraction has occurred. If the cavity is actively secreting, the diagnosis may be difficult if the expectoration contains nothing characteristic, and the signs may resemble those of tuberculosis, abscess, or gangrene of the lungs.

Sudden and severe pain and dyspnœa with signs of air and fluid in the pleural cavity characterize rupture into that cavity. Rupture into the pericardial sac may be immediately fatal. If not, there will be increased præcordial dullness with diminished or absent cardiac impulse. A cyst of the liver projecting into the lung may rupture into a bronchus, in which case the expectoration will be stained with bile. When cysts of the right lobe of the liver project upward it may be difficult to determine whether the cyst is above or below the diaphragm. Pressure effects on the structures within the chest are not so common with hydatid cysts as with other forms of tumors. If fluctuation is present and pleural effusion can be excluded, the fluctuation is most likely due to an hydatid cyst.

TREATMENT.—The medical treatment of hydatids of the lungs is symptomatic only. Surgical measures alone can hope to effect a cure. While paracentesis has resulted in a cure in a few instances, it is regarded by most observers as a dangerous procedure, as the fluid may escape into the bronchi and cause a fatal result. The same surgical course should be pursued as in dealing with a pulmonary abscess or other lung cavity. The question of whether to simply drain the cavity, or to remove also the ectocyst, is one which must be decided by the conditions of the tissues and the surgeon's experience.

TUMORS OF THE LUNGS.

Tumors of the lungs may be primary or secondary. Primary growths are rare in the lungs. Secondary growths are more common. [In 112 cases of tumors of the lungs the disease was primary in 38, and secondary in 64—Wilson Fox.] The primary growths are usually epithelioma, encephaloid, or scirrhus, the first named being the most common. Sarcoma may occasionally be found in the lungs as a primary growth, and enchondroma very rarely occurs as such. Primary cancerous growths may originate in the glands of the bronchi, or in the alveolar epithelium. They may spread along the lymph channels.

Sarcomata are most often secondary though they may be primary. The most frequent variety is the round-cell form, though spindle-celled, myeloid, melanotic, and osteo-sarcomatous forms are also met with.

The secondary growths may be encephaloid, scirrhus, epithelioma, colloid, enchondroma, melano-sarcoma, or osteoma. Enchondroma may occur in the bronchial cartilages.

ÆTIOLOGY.—Tumor of the lungs is most common between the fortieth and sixtieth years of life. The primary form affects the sexes equally (Walshe). The secondary form is most common in women. It is not clear that the fact pointed out by Williams that in cancerous disease generally there is a family history of tuberculosis rather than one of malignant disease, will apply to primary malignant disease of the lungs. It is claimed that a large proportion of the deaths of

persons over forty years of age who are employed in the cobalt mines of Schneeberg, is due to malignant disease of the lungs.

The secondary growths usually follow disease of the genito-urinary system, digestive tract, or of the bones.

MORBID ANATOMY.—Primary carcinoma and sarcoma usually occur in but one lung and may form large masses involving the greater portion of the lung. Secondary growths are usually distributed through both lungs though they may be localized and involve chiefly the pleura. Metastatic growths are usually disseminated and may involve large portions of the lungs. Primary growths when limited to a single lung generally affect the right lung. Secondary multiple growths usually affect both lungs alike.

Primary growths may occur as well defined tumors or as infiltrations, the latter being most common. All traces of lung tissue may be destroyed. The growth may extend into the lung tissue along the perivascular and peribronchial lymph spaces. Cancerous growths may be soft and fleshy in appearance, or may be hard, solid, white masses. The site of the bronchial glands may be marked by pigmentation.

Sarcomata may form masses an inch or two in diameter, or there may be numerous nodules scattered through the lung tissue. They are well defined, soft, and have a fleshy appearance. The sub-pleural and pulmonary lymphatics are usually infiltrated.

Secondary growths usually form small, multiple, disseminated nodules in both lungs. They may resemble tubercles. Large, single masses may be present.

With widely disseminated growths or with dense infiltrations the lungs may be enlarged, though some retraction may at times be present. Softening, excoriation, and pus collections may occur. Cancerous, sero-fibrinous, or hæmorrhagic pleurisy may be an associated lesion. The mediastinal and bronchial glands are usually affected, less frequently the cervical glands are involved, and rarely the inguinal glands are enlarged.

CLINICAL HISTORY.—The clinical history of tumors of the lungs may be very indefinite, especially with primary growths. There may be no symptoms of a lesion in the chest

(Walshe). Pain may be an important symptom if the pleura is involved. Dyspnœa may be troublesome and may be paroxysmal if there is pressure on the trachea. Dry, painful cough, mucoid expectoration may be present. Stokes has called attention to "prune-juice" expectoration in some cases of primary cancer of the lung. Passive congestion of the face and of one or both upper extremities may occur from pressure. The pneumogastric or recurrent laryngeal nerves may show pressure effects. If the mediastinum is not involved the pressure effects will be absent. The rapid dissemination of the lung lesions may be attended with pneumonic symptoms and albuminuria (Wunderlich). Emaciation may or may not be considerable.

The duration of the disease is from six to eight months. It may run a very acute course. Jaccoud observed a case which lasted only a week from the onset of the symptoms.

SYMPTOMS AND DIAGNOSIS.—Primary growths not involving the mediastinum are so rare, and, pressure effects being absent, their symptoms so indefinite, that they are often not recognized when present. When an intrapulmonary growth is large there may be some bulging of the side, though retraction may be present from extensive infiltration or from collapse due to bronchial obstruction. The heart may be displaced. Vocal fremitus may be normal, increased, or absent. Pulsation may occur from transmitted heart's action. The percussion note may present any quality from normality to absolute dullness. Extension of dullness beyond the median line is as important, but of less common occurrence, as in mediastinal tumors. The breath-sounds are usually diminished in intensity. Bronchial breathing may be present.

The diagnosis is not difficult in secondary growths, as the history of the patient will be suggestive. Progressive emaciation, involvement of the cervical glands, especially the clavicular, dark-colored expectoration, and anomalous physical signs will suggest the nature of the trouble.

TREATMENT.—The treatment of tumors of the lung is symptomatic. In special cases it is possible that surgical measures might be considered with advantage.

CHAPTER XVI.

PLEURISY.

Inflammation of the pleura may be primary or secondary. As to its cause it may be acute or chronic. From an anatomical point pleurisy may be divided into dry pleurisy, fibrinous, plastic or adhesive pleurisy, and pleurisy with effusion. According to the character of the effusion pleurisy is spoken of as serous, sero-fibrinous, sero-purulent, purulent (empyema), hæmorrhagic, and putrid. The terms traumatic, diaphragmatic or phrenic, cancerous, tubercular, etc., are sometimes used to indicate special features of individual cases. Attempts have been made to classify pleurisy from a bacteriological standpoint, and there has been described a pneumococcus pleurisy, a streptococcus pleurisy, a saprogenic pleurisy, a staphylococcus pleurisy, a tubercular pleurisy, and pleurisy due to such organisms as the typhoid bacillus, the influenza bacillus etc.

While in many instances of tubercular and of pneumococcus infection of the pleura a characteristic inflammation results which can be defined clinically, in the majority of cases the part played by the various infections cannot, as yet, be outlined clinically with sufficient clearness to make such a classification of practical utility. Regarding the disease purely from a clinical standpoint the simplest classification is one under which may be included the greatest number of cases without the subdivision which is necessary when we attempt an ætiological classification. Dividing pleurisy, then, into fibrinous or plastic pleurisy, sero-fibrinous pleurisy, and purulent pleurisy, we have a classification embracing the clinical features of the majority of cases and which is more definite than the simple division into acute and chronic pleurisy, and which is less elaborate and confusing than a classification based on the numerous ætiological factors of the inflammations of the pleura.

Fibrinous (plastic, adhesive) pleurisy may be primary or secondary, acute or chronic (primitive dry pleurisy). It may occur in any degree from the slight inflammation with little or no exudation (dry pleurisy), to extensive plastic exudations resulting in considerable induration of the pleuræ. It is probable that the pleuritic adhesions so frequently found post mortem in subjects in whom there has been no history of pleural disease are the result of the milder degrees of fibrinous pleurisy.

ÆTIOLOGY.—Primary, fibrinous pleurisy may occur as an independent affection, in persons of good health, from exposure to cold. It is not clearly understood just how the disease is thus brought about, though we have bacteriological evidence of the loss of resistance in tissues after exposure to cold, and we know that individual resistance in this respect varies greatly. It is not improbable that a clearer appreciation of the practical workings of pneumo-dynamics might explain some of this class of cases. All constitutional conditions which lower the vitality of the individual may predispose toward plastic inflammation of the pleura.

Some cases of primary, plastic pleurisy are undoubtedly tubercular in origin. The exact proportion is difficult to estimate. In general serous-membrane tuberculosis the pleura may be involved simultaneously with the peritoneum. In tubercular pleurisy the visceral pleura is infected through the sub-pleural lymphatics or from the bronchial or tracheal lymph glands. The parietal pleura may be infected from the cervical, vertebral, or mediastinal lymph glands, or through the diaphragm from the lymphatics of the peritoneum. The frequent impossibility of demonstrating the tubercular nature of the adhesions which are often present in cases of pulmonary tuberculosis shows the difficulty of recognizing the tubercular nature of a primary pleurisy. While the adhesions which are associated with pulmonary tuberculosis may be due, in some instances, to associated infections, it is altogether likely that in the great majority of cases they are tubercular in origin. This latter is more readily demonstrated, according to Fowler, by examination of the pleural surfaces of the interlobar fissures where the tubercular granulations are most distinct.

Secondary, fibrinous pleurisy ocurs in connection with acute diseases of the lungs such as pneumonia, tuberculosis, cancer, abscess, gangrene; to inflammations of the pericardium and mediastinum. Bright's disease, and acute rheumatism may precede this form of pleurisy. Fractures of the ribs, injuries of the chest walls, gun-shot and other perforating wounds of the pleura may cause plastic pleurisy when the injury is not septic in character.

Morbid anatomy.—In fibrinous pleurisy there is a fibrinous exudate upon the pleural surface. Leucocytes and blood corpuscles are enclosed in the meshes of this exudation. Preceding the appearance of this fibrinous exudate there is injection of the subserous vessels, loss of lustre of the membrane, proliferation of the nuclei of the endothelial cells of the pleura, and blocking of the stomata of the membrane and of the adjacent lymphatics with fibrin and corpuscles. The layer of coagulable lymph varies in thickness with the amount of cellular elements and fibrin poured out. The layers of lymph nearest the pleural membrane may be dense and laminated; superficially the lymph is trabeculated with a fine net work of fibrin enclosing corpuscles. If the inflammatory process ceases at this point (dry pleurisy) the exudation may become vascularised and form connective tissue with more or less union of the surfaces of the pleura. The exudation may become absorbed without the formation of adhesions, and some thickening and opacity of the pleura may be the only result of its presence. Thickening of the interlobular septa is a frequent result. In chronic tubercular pleurisies of this type the layers of exudation may be very thick and laminated and may be granular in appearance from prolonged friction of the surfaces of the pleura. In tubercular pleurisy particularly, the pleural membrane lining the interlobar fissures is frequently much thickened and closely adherent. In the chronic forms of fibrinous pleurisy great induration of the visceral pleura may result, and the adjacent interlobular lung tissue may be involved. In fibrinous pleurisy the visceral pleura is much more frequently involved than the parietal pleura owing to the frequency of the occurrence of pleurisy secondarily to inflammations of the lung. The lower portion of the

pleura is much more frequently affected than the upper espe-
cially in tuberculous cases, though in apical tuberculosis of
the lung the pleura is affected more often than has been sup-
posed. In some cases the inflammation is limited to the
diaphragmatic pleura (diaphragmatic pleurisy), and though
any variety of pleurisy may be limited to the diaphragmatic
pleura, such an inflammation is usually of a fibrinous nature.

CLINICAL HISTORY.—The clinical history of acute plastic
pleurisy is usually distinctive. Pain in the side is the earliest
and most prominent symptom. Preceding or accompanying
the onset of the pain there may or may not have been chilliness
or a chill. If the latter occurs it is usually of a nervous char-
acter. The pain is sharp and piercing, and usually begins very
suddenly at about the middle of inspiration and ceases with
the beginning of expiration. In some cases, especially in the
apical pleurisies accompanying pneumonia of the same region
in children, the pain may be more or less continuous.

Dry, hacking cough is present. It is apt to be constant
and causes considerable pain. Dyspnœa, if present, is irregu-
lar and results largely from fear of pain. The temperature
is usually elevated two or three degrees. The pulse is rapid
(from 100–120) quick, small and irregular in force. In some
cases of limited inflammation there may be no history except
that of slight cough, slight pain on deep inspiration, and a
slight febrile rise. In pleurisy secondary to other diseases of
the chest the occurrence of the above symptoms will be readily
understood though they be masked by the symptoms of the
original disease. The majority of cases of fibrinous pleurisy
which are observed in this stage are not followed by effusion;
the symptoms subside within two weeks and the patient is
apparently as well as ever. If the inflammation has been ex-
tensive, recovery may be slow and there may be more or less
thickening of the pleura with permanent loss of expansion of
the affected side. In some of these cases of acute plastic pleu-
risy, liquid effusion will result, or rather the plastic exudation
and the sero-fibrinous effusion are practically simultaneous in
their appearance, as in the following case:

Man aged thirty-five years. Three days ago developed a sharp pain in right side. Pain has continued ever since and is still severe when he coughs or breathes deeply. Temperature 101°; pulse 100. Examination shows loss of motion of lower right side. Heart apex displaced one inch to the left. Fremitus absent below sixth rib on right side. Partial dullness on right side between fifth and sixth interspaces; flatness below sixth rib. Well marked friction sound from fifth to seventh ribs on right side. Exploratory puncture in seventh interspace obtains fluid of a turbid character containing flakes of lymph.

In this case the plastic exudate was considerable, and the liquid effusion moderate in amount, as sufficient time had not elapsed, in all probability, for the effusion to reach its extent. The friction sound, which is usually absent when sufficient fluid is present to be readily recognized, was therefore well marked.

Primary, plastic pleurisy may result in great thickening of the pleural membranes and result in cirrhotic processes in the lungs (Sir Andrew Clark). This condition is clinically identical with like conditions which result from chronic empyema, long standing encysted serous effusions, or primary pulmonary cirrhosis, and its occurrence as a pathological or clinical entity has been denied.

In primary, plastic pleurisy of tubercular origin there may be great thickening of both pleural layers, eventually much retraction of the side, and a history of "stitch in the side," moderate cough, slight fever, gradual loss of strength, etc., extending over a period of a year or two.

The following case was of this nature:

Young woman twenty-two years old. Sick eleven months, previous health good. Family history good except as to an aunt, who died of phthisis. About a year ago patient began to have pain in the left side extending from the nipple line around to the point of the scapula. This was followed by slight cough, gradual loss of flesh, and for the last two months, amenorrhœa. Her temperature was 99.8°; pulse 100.

The left side shows slight retraction below the level of the fifth rib, considerable loss of expansion over same area with inspiratory recession of the interspaces. Deep inspiration causes pain and cough. Fremitus, pitch of voice and inspiratory sounds, pitch of percussion note all markedly increased over this area. Very little air enters the lower portion of the left lung. No bacilli could be found in the sputum until within the last two months. During this period, however, there have been signs of consolidation and catarrh of the left apex.

SYMPTOMS AND DIAGNOSIS.—The pain of acute, plastic pleurisy is severe and lancinating. It usually catches the patient suddenly at about the middle of the inspiratory act. It is ordinarily felt in the axillary region or just below the nipple at the level of the fourth or fifth interspace. In secondary, apical pleurisy the pain is felt at the top of the shoulder and beneath the upper portion of the scapula. In diaphragmatic pleurisy the pain may be felt lower down in the back or in the abdomen and pressure over the diaphragmatic insertion at the tenth rib increases the pain. The pain of pleurisy is increased by deep breathing and by coughing. The patient adjusts his position so as to limit the motion of the affected side, usually by flexing his body, leaning toward the affected side, and clasping the ribs by one or both hands when he coughs. His face has an anxious, pained expression. There is moderate elevation of temperature and a quickened pulse. The cough is spasmodic, hacking and painful. Expectoration is scanty or absent.

The respiration is jerky, and the motion of the affected side is diminished in acute cases largely from fear of pain though in chronic cases there may be much loss of expansion, and even retraction of the chest wall, from thickness and induration of the pleural and sub-pleural tissues. The fremitus is unchanged in acute cases. In chronic cases it may be increased over the lower portion of the chest wall. The percussion note is usually not perceptibly altered unless there has been considerable plastic exudate when it may be slightly dull.

On auscultation we obtain the only definite sign of this

variety of pleurisy, i. e., the friction sound. Pleural friction sounds may be grazing, rubbing, or grating in character. They are heard about the middle of the inspiratory act and cease before the inspiration is completed. The grazing or fine crepitating sounds are obtained in the earliest stage before much exudation is present. They may last but a short time. They may closely resemble the crepitating rale of pneumonia, but occur earlier in the inspiratory act and are distributed over a longer period of time than the latter. The sounds are also not so distinctly individual as are the numerous, bunched crepitations of pneumonia. If there is moderate plastic exudate the sounds become rubbing or grating in character. If considerable exudation occurs the friction sound disappears. If there is fluid effusion the sounds may disappear early if the effusion is abundant and they may reappear when the fluid is absorbed in which event they will be dry and rubbing in character (reduction rales). With considerable plastic exudate and liquid effusion of moderate amount the friction sounds may remain throughout the course of the disease. If the exudation is entirely plastic the friction sounds gradually become rubbing in character and more dry in quality and disappear within a week or two.

The voice sounds and respiratory murmur are usually diminished except in chronic cases with thickened pleura and adhesions. They will then be raised in pitch but diminished in volume. Loss of expansion, inspiratory recession of intercostal spaces, increased tactile fremitus, diminution in volume and increase in pitch of the vocal and respiratory sounds may indicate pleural adhesions, but, as these symptoms may be present without adhesions, the latter can only be diagnosed provisionally, seldom positively.

In diaphragmatic pleurisy the respiration is usually short, and is thoracic in type. The motion of the diaphragm is limited. Severe dyspnœa may be present with attacks resembling angina (Andral). Objectively it is difficult to explain the severity of the subjective symptoms of diaphragmatic pleurisy.

The diagnosis of acute plastic pleurisy is usually not difficult. In interlobar pleurisy if there are encysted collections of fluid the diagnosis may be difficult. In some instances the

onset of plastic pleurisy may resemble that of pneumonia to a marked degree with the exception of the relation of the pulse and respiration rate which is less distinctive than in pneumonia.

The following case illustrates this type of the onset of pleurisy:

Man aged 45, large and fleshy. Previous health good. Was taken ill suddenly with a chill followed by fever and pain in the side, cough, slight expectoration, and dyspnœa. Temperature 102.8°, pulse 100, respiration 32. On examination there were bronchial rales in the left lung; no consolidation. Over the lower portion of the left pleura, anteriorly, there was a very rough, grating friction sound. In twenty-four hours the temperature came down to 100.5° and the cough and dyspnœa were relieved. The friction sound became less rough and disappeared in five or six days.

The pain of myalgia or of intercostal neuralgia may simulate that of pleurisy but the tender spots may be definitely outlined and friction sounds are absent. The chronic types of fibrinous pleurisy may, especially when tubercular in nature, furnish the signs of encysted collections of fluid, and the only definite means of differentiation may be the use of the exploring needle.

TREATMENT.—The treatment of fibrinous pleurisy is largely symptomatic. For the pain the most efficient remedy is one-fourth of a grain of morphia hypodermically. In diaphragmatic pleurisy morphia is generally a necessity. Next to morphine the quickest relief is afforded by a good-sized fly blister. When these means are objectionable, especially in children, the hot poultice, mustard plaster, or hot applications are very useful. Blisters, while relieving pain, seem, also, to have some modifying action on the intensity of the inflammatory process, and their application is, therefore, good treatment in cases of active inflammation. A single blister, 4x5 or 5x6, is better than several, or a succession of, small ones. Leeches, cupping, cauterization, etc., are troublesome and inefficient methods compared with the above treatment. Leeches are better than cupping for the relief of pain.

Fixing the side by means of long strips of adhesive plas-
ter passing over the median line and drawn tightly is highly
recommended by Roberts and others. It certainly gives much
relief. The ice bag is recommended, but does not seem to be
of much service. The fever and cough should be treated
symptomatically. The bowels should be managed by a little
calomel followed by a saline. The use of large doses of salicy-
late of soda has been recommended for the limitation of the
inflammation and the prevention of liquid effusion, but they
do not seem to have such effects and are apparently useless.
Iron, quinine and strychnine are very useful in overcoming
the depression which follows the attack.

SERO-FIBRINOUS PLEURISY.

Sero-fibrinous effusion occurs in pleural inflammation of
a somewhat more severe character than that in which fibrinous
exudation alone occurs. This form of pleurisy occurs at any
time of life, though most common in adults. Men are more
frequently affected than women.

ÆTIOLOGY.—All the causes of acute plastic pleurisy are
factors in the production of the sero-fibrinous variety, as more
or less plastic exudation precedes or accompanies sero-
fibrinous effusions. Rheumatism, Bright's disease, inflamma-
tions of the lungs, pericardium, or mediastinum may be ac-
companied by sero-fibrinous pleurisy. A large proportion of
the cases of this variety of pleurisy belong to the so-called
idiopathic pleurisies. Bacteriological research has shown that
most of these cases are due to certain organisms. The bacillus
tuberculosis is the organism most frequently causing sero-
fibrinous pleurisy, while in some few instances the pneumo-
coccus appears to be the causative agent. Coverslip prepara-
tions, cultures and ordinary inoculation tests may fail to show
the tubercle bacillus as they are often very few in number.
Eichorst found that by injecting 15 cc. of fluid exudate into
test animals 62 per cent. of the cases were shown to be tuber-
culous. Le Damany, by injecting large quantities of fluid,
demonstrated all but four of fifty-five cases to be tuberculous.
Bowditch traced the history of ninety cases occurring between

1849 and 1879, and found that thirty had died of tuberculosis. Of 101 cases of fibrinous, sero-fibrinous, hæmorrhagic and purulent pleurisy, Osler found thirty-two cases to be definitely tubercular. From the statements of French authors it would appear that from 70 to 75 per cent. of the cases of acute pleurisy are tuberculous. Observations of series of cases by Kelsch and Vallard, Lemoine, Netter, Chaufford and Gombault, Thue, Fiedler, Barrs, Richochon and others have shown, either through inoculation experiments or through the subsequent history of the cases, that from one-third to one-half of the cases were tubercular. Staphylococci, streptococci, pneumococci, Eberth's typhoid bacillus and other micrococci have been found in the effusion of pleurisy by Thue, Fernet, Sidney Martin and others.

The clinical fact that pleurisy often will rapidly follow a sudden exposure, wetting, or chill is not denied by those who are the strongest advocates of the growing belief that these cases are, as Sée expresses it, "tubercular pleurisy, the nature of which has been misunderstood." We cannot, at present ascribe all sero-fibrinous pleurisies to microbic infection, but as far as their tubercular nature is concerned, the following may be accepted: That a considerable proportion of the cases which come on suddenly in healthy people are of a tuberculous nature; that a somewhat larger proportion of the sub-acute or insidiously appearing cases in persons whose present health is not of the best are tuberculous in nature; and that sero-fibrinous pleurisies secondary to chronic diseases are also frequently tubercular in origin.

The difficulty of proving the tubercular nature of some cases, which eventually exhibit their tubercular nature by examination of the fluid or by inoculation experiments, causes the subsequent history of these cases to be of special interest. The fact that many cases recover and never exhibit any evidence of tuberculosis is rather an argument in favor of the curability of this variety of tuberculosis of the pleural, serous membrane than an indication of its non-tubercular nature. Yet it is difficult to exclude the doubtful element of subsequent infection in those cases where considerable time has intervened between the recovery from the pleural inflammation and the

appearance of recognizable tuberculosis. We must know something of the liability to subsequent infection, the character of the morbid process, and the nature of its onset in the individual cases before we can estimate the value of statistics bearing on the fact of the subsequent development of tuberculosis in persons with a previous history of pleural inflammation.

It is a perfectly safe clinical rule to regard persons who have had sero-fibrinous pleural effusions, especially when such effusions come on insidiously without evident cause in persons of an indifferent state of health, as candidates for tuberculosis, and to frame our advice and medication on that basis.

Sero-fibrinous effusions may occur after pneumonia though such occurrence is rare, meta-pneumonic pleurisy being usually purulent. Sero-fibrinous effusions may occur in the free pleural cavity adjacent to encysted purulent effusions.

MORBID ANATOMY.—In sero-fibrinous pleurisy the proportion of serum and fibrin varies greatly. The fibrin will be found on the surfaces of the pleura or may be floating as floculi in the liquid effusion. It may be very scanty in amount or may form thick layers or masses. It may deposit as thick, curdy masses or layers at the most dependent portion of the pleural cavity. The liquid effusion may be clear, or slightly turbid; lemon-yellow, greenish, dark brown or red (hæmorrhagic pleurisy) in color. The usual color is light yellow with a greenish tinge. The fluid is alkaline in reaction and has a specific gravity of from 1.005 to 1.030 (Fraentzel). It contains leucocytes, large cells probably derived from the pleural endothelium, red blood corpuscles, and shreds of fibrin. If the corpuscular elements are few, the fluid is clear and translucent; if they are numerous, the fluid is opalescent or turbid. Urea, uric acid, sugar, cholesterine, leucine, tyrosin and xanthoxine may also be found in the fluid. The fluid is albuminous, the amount of albumen varying from 31 to 77 per cent. (Walsh). After withdrawal of the fluid it may coagulate spontaneously. Hæmorrhagic effusion is rare. It may occur in connection with cancer, tuberculosis, Bright's disease, cirrhosis of the liver, with malignant types of specific fevers, or independently of any of these affections. The quantity of

fluid present varies from a few ounces to several pints. The fluid may be loculated or encysted in small sacs by means of masses of lymph or by adhesions. These small collections of fluid usually occur in the lower portions of the pleural cavity, but may occur in any part. One sac, or portion of the pleural cavity, may contain serous effusion, and at the same time another portion of the pleural cavity may be occupied by a purulent collection of fluid. The displacement of the organs within the chest which results from effusion is readily reduced upon removal of the fluid unless prevented by extensive deposition of lymph or failure of the collapsed lung to expand.

In large effusions the heart and mediastinum are displaced toward the sound side unless retained by adhesions. This occurs early and does not represent the degree of positive pressure within the pleura as closely as does the downward displacement of the diaphragm or liver. The apex of the heart may be raised and the heart may lie more transversely, in left-sided effusions. In effusions of the right side the heart is displaced to the left without much or any change in level. The heart does not rotate on its axis in these displacements. A large portion, or the whole, of the lung may be collapsed, airless, bloodless, grayish in color and lie close to the spine.

The displacement of the heart is due in part to the loss of the usual elastic traction of the lung of the affected side and may be present in small localized collections of fluid which could not possibly exert much pressure on the heart. The collapse of the lung is likewise due in part to the loss of expansion and tractile force in the lung of the affected side and to the elasticity of the lung tissue which causes it to retract, as the fluid accumulating in the pleural cavity alters the state of the intra-pleural tension, and as the air remaining in the alveoli is absorbed. With small effusions, especially localized ones occupying the lower portion of the pleural cavity, that portion of the lower lobe in contact with the effusion is usually collapsed. When these collections occur in the anterior part of the left pleura the heart may be displaced upward and inward without there being any other marked evidence of pleurisy. Thus, in a young man this displacement of the heart

led to the discovery of a localized collection of fluid in the
lower anterior part of the left pleural cavity, and from which
the withdrawal of four ounces of fluid was followed by a
reposition of the heart to its normal situation.

CLINICAL HISTORY.—The clinical history of sero-fibrin-
ous pleurisy varies with the nature of the case. Prodromal
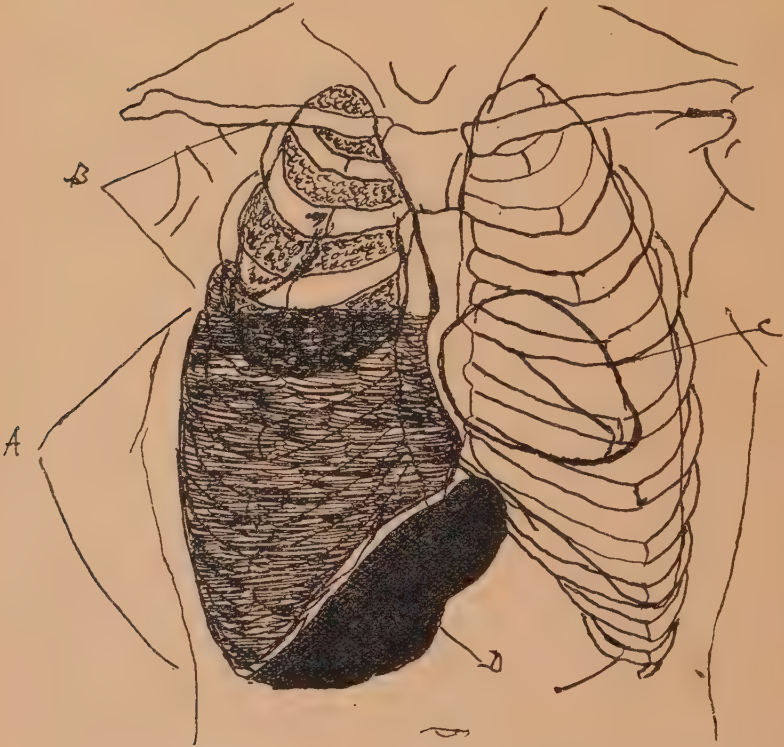
Displacement of organs in a considerable effusion into right pleura.
A.—Slight enlargement of side, with effacement of intercostal
spaces. B.—Compressed lung. C.—Displaced heart. D.—Displaced
liver.

symptoms may or may not be present. In some instances the
onset is abrupt, with chill, fever, and pain in the side,—the
manifestations of acute plastic pleurisy. These are followed
by a more or less rapidly accumulating fluid effusion. The
fever may reach 102° or 103° at first and drop a degree or so

when the effusion is established. In cases where plastic exudation and sero-fibrinous effusion are both well marked we will get pronounced subjective symptoms of the former and definite objective evidences of the latter.

In many cases with considerable effusion the onset is insidious, and there is an entire absence of definite history, the patient going about his usual avocations, if they do not entail physical labor, without great difficulty, suffering only slight shortness of breath, some cough, and general weakness.

A physician recently presented himself, stating that he "did not feel quite right," and thought he would come in and see what was the matter. He was attending to a fairly large general practice without special difficulty except for feeling weak and having some shortness of breath when he hurried. He had a slight cough, and his temperature was 100.5°. His left pleural cavity contained over three pints of fluid.

A boy sixteen years old came into the clinic having arrived in Chicago two days previously from Philadelphia. He complained of feeling weak during the journey and was short of breath after his arrival. His previous health had been good. His temperature was 101°. Forty-two ounces of fluid was withdrawn from his left pleural cavity.

In this class of cases the fluid may accumulate quite rapidly.

An office patient complained of slight cough, general weakness, and exhibited two and a half degrees of fever. Examination of his chest determined nothing of special interest. He returned two days later no better. A second examination was negative as far as his pleuræ were concerned. Two days later he returned in the same condition, and as he was a complaining and loquacious individual he was prescribed for and dismissed without further examination. The next day he consulted another physician who withdrew over three pints of fluid from his chest.

In the so-called "latent" type of pleurisy, especially when tubercular in nature, there may be simply a history of slight cough, slight dyspnœa, and a low grade of fever continuing for long periods. There may be slight retraction of the side. In other cases, especially of encysted tubercular effusions, there may be a febrile history resembling mild remittent malarial fever, or irregular typhoid. In a recent case of this kind in a woman who had exhibited an irregular fever for four or five weeks, the removal of a few ounces of fluid from an encysted effusion in the left pleural cavity resulted in immediate improvement.

When sero-fibrinous effusions are associated with encysted purulent effusion the history of the latter will predominate, and the diagnosis of the former will rest entirely on the physical examination. The persistence of fever and rapid pulse after thorough drainage of the empyema, may serve to direct attention to the serous effusion, as in the following case:

Man aged 35. Encysted empyema in right pleural cavity. Excision of a portion of the seventh rib and drainage. Pus cavity well contracted, small and well drained. Temperature and pulse remained high notwithstanding the good drainage. Palpation of the cavity showed it to be completely walled off from the remainder of the pleural cavity by dense adhesions. Examination of the upper portion of the right chest showed evidences of fluid above the fifth interspace—the upper boundary of the wall of adhesions surrounding the drained cavity. Puncture in the third interspace obtained sero-fibrinous fluid, about a pint of which was withdrawn. The temperature and pulse improved but the patient died from general sepsis.

The course of sero-fibrinous pleurisy is variable. In acute cases the fever may disappear in a week and slight or moderate effusions may be absorbed. In others the effusion gradually increases, the pain and fever continuing for from twelve to sixteen days when improvement will begin. In the sub-acute cases when the effusion reaches as high as the third rib recovery will be slow. These cases may have lasted two or three weeks before coming under observation, and while the

natural tendency is toward absorption this may not take place until a portion of the fluid is removed by aspiration or otherwise. Absorption is then slow and is followed by rough friction crepitus when the pleural surfaces come in contact. As a rule there is loss of resonance, and feeble breathing over the base of the lung for a considerable period.

A sero-fibrinous effusion may last for months without change, particularly in encysted tubercular pleurisy. Loculated effusions consisting of a pint or so of fluid are more apt to recur than are small effusions of from four to eight ounces. Large free effusions are not so apt to re-accumulate though they do so in a small proportion of cases. In some cases effusions will recur repeatedly in spite of aspiration and all other treatment. Cases of perforation and discharge of the effusion through the lung or chest wall have been reported (Sahli, Harris).

Sero-fibrinous effusion, when not treated, may gradually increase for one or two weeks. There is then a stationary period lasting a few days which is followed by gradual absorption. Some cases of large effusions may disappear rapidly, and, again, they may last for months without a change. Spontaneous absorption rarely takes place during the period of marked fever, though this may occur in tuberculous cases. The moderate temperature of sub-acute cases usually disappears soon after aspiration. In acute cases the fever is slower to disappear after aspiration.

When a pleural cavity is full of fluid and the heart is greatly displaced, there will be, as a rule, comparatively little disturbance. There may, however, be danger of sudden death from syncope caused by some slight exertion. According to Weil, fatal syncope may be due to thrombosis or embolism of the heart or pulmonary artery, œdema of the opposite lung, or to degeneration of the heart muscle. He thinks that the mechanical effect of the displacement of the heart, or of the twisting of the great vessels, in the production of syncope, is not clearly demonstrated. Sudden death has in some instances been preceded by paroxysmal attacks of dyspnœa, or attacks of syncope. In some cases sudden death occurs without premonitory symptoms.

SYMPTOMS AND DIAGNOSIS.—The subjective symptoms of sero-fibrinous pleurisy may be very variable as shown by the clinical history already detailed. Moderate fever, dyspnœa, slight cough, pain and weakness are the chief complaints. The fever range varies greatly with the nature of the case. Usually it is not a prominent symptom. The pain usually ceases when the effusion becomes considerable, but may return to some extent when absorption is effected. In cases of slight effusion and considerable plastic exudation the pain may be continued during the course of the disease. The cough is usually a characteristically short, hacking affair to which little attention is paid. Dyspnœa is troublesome on exertion. There may be a tendency to attacks of syncope. In sub-acute cases with large effusions the weakness, dyspnœa on exertion, and short, dry cough are the symptoms most likely to be remarked on by the patient in describing his condition.

The physical signs of effusion within the pleural cavity may or may not be determinate and conclusive. Large effusions are usually recognized with ease, but small effusions, or loculated ones, may be very difficult to determine by the physical signs alone. Thickening of the visceral pleura from induration subsequent to the deposit of quantities of exudate in acute plastic pleurisy, and consecutive interstitial fibrosis of the lung with partial collapse may furnish conditions which produce physical signs in every way similar to those of encapsulated effusion of moderate dimensions.

Inspection is important in effusions of any extent. If there is a large effusion the shape of the chest may be altered, the affected side appearing larger and its outline rounded. The depressions of the intercostal spaces may be effaced, or, rarely, the intercostal spaces may be bulging. (According to Stokes, bulging of the intercostal spaces is due to inflammatory paralysis rather than to intra-thoracic pressure alone, and indicates a severe lesion.) Measurements and cyrtometer tracings show that the enlargement of the side is more apparent than real, though according to Powell the total circumference of the chest is always increased. The patient may lean toward the affected side and there may be an evident degre of scoliosis. Extensive effusions in the right side

may cause a fullness of the side from displacement of the liver. In small, loculated effusions in the lower portion of the pleural cavity the chest wall may be retracted and measure less than the opposite side and there may be depression and inspiratory retraction of the interspaces.

In large effusions the loss of respiratory excursion on the affected side is at once apparent. Expansion of the affected side may be limited to the upper portion, or the whole side may appear motionless. In encapsulated effusions the loss of motion is limited to the area affected, generally the lower portion of the chest.

Cardiac displacement, as shown by dislocation of the apex beat, is one of the most reliable evidences of pleural effusion. In effusion on the right side the displacement is less, relative to the amount of effusion, than in left effusions. In right effusions the apex is displaced to the left and slightly upwards, and may be displaced as far as the anterior axillary line. In left effusions if the apex is underneath the sternum it may not be discernible. It may be displaced to the right as far as the right nipple line. I entirely agree with Powell in his view as to the importance of cardiac displacement as an early and constant sign of pleural effusion, but not with his statement that it occurs *pari passu* with the extent of the effusion. In this respect the degree of displacement is not to be regarded as strictly related to the amount of effusion in all instances. There are pneumo-dynamic forces other than positive increase in intrapleural tension which cause the cardiac displacement—such as the elastic traction force of the opposite lung—hence, as in cases already cited, we may get displacement of the heart with very small encapsulated effusions. This absence of ratio between the extent of the effusion and degree of displacement of the heart renders the latter all the more valuable as a sign of pleural effusion, as displacement of the diaphragm, liver, and other organs which is dependent on positive intrapleural tension are late signs of effusion and only obtained, as a rule, in large effusions and where other positive signs have been previously obtained.

By palpation we can at times more clearly determine the variations in chest expansion, the alteration in the intercostal

spaces, or the displacement of the apex beat, than we can by inspection. We have, besides, absence of vocal fremitus over the area occupied by the fluid. This is the rule, though in small effusions where there are adhesions and especially in encapsulated effusions, the fremitus may be present or even increased if the lung has not collapsed and has undergone a moderate degree of fibrosis. It is very rare that fluctuation can be detected. Pulsation may very rarely be felt in sero-fibrinous effusions. Œdema of the chest wall is seldom found. In children vocal fremitus may be present over the area of effusion in the absence of any adhesions.

Percussion affords us valuable evidence of the presence of effusion. Percussion over the area of fluid gives a peculiarly toneless, flat note. The lack of tone is distinctive, though in small or loculated effusions the note may be modified to such an extent that it differs but little from that given forth by collapsed or extensively indurated lung tissue.

The limits of the dullness are important and are best determined by light percussion. In encapsulated effusions the limits of dullness will have no distinctive features. In moderate sized or large, free effusions the upper and lower borders are important. The upper border of flatness may be found at any elevation according to the amount of effusion. With moderate effusion, no adhesions, and an elastic lung the line of flatness may be lower in the axillary and mammary regions when the patient is lying down than when sitting or standing. This is a positive evidence of fluid, but it is seldom that it can be definitely determined. With considerable effusion the upper border of flatness is irregular and has been described as Damoiseau's curve, or the letter S curved line of Ellis. It is highest in the axillary and scapular regions and dips downward in the interscapular region leaving a tongue of comparative dullness in the postero-median line which Garland has called the "dull triangle." Anteriorly the line declines gradually until it reaches the sternum. According to Garland the line always begins at a lower level behind than in front. In some cases of considerable effusion this line can be readily made out, in many it cannot be definitely determined. The lower border of flatness is outlined by the curved line of the

diaphragmatic arch on the left side, while on the right the
flatness runs into that of the liver. In large effusions of the
left side the lower line of flatness may be straightened and
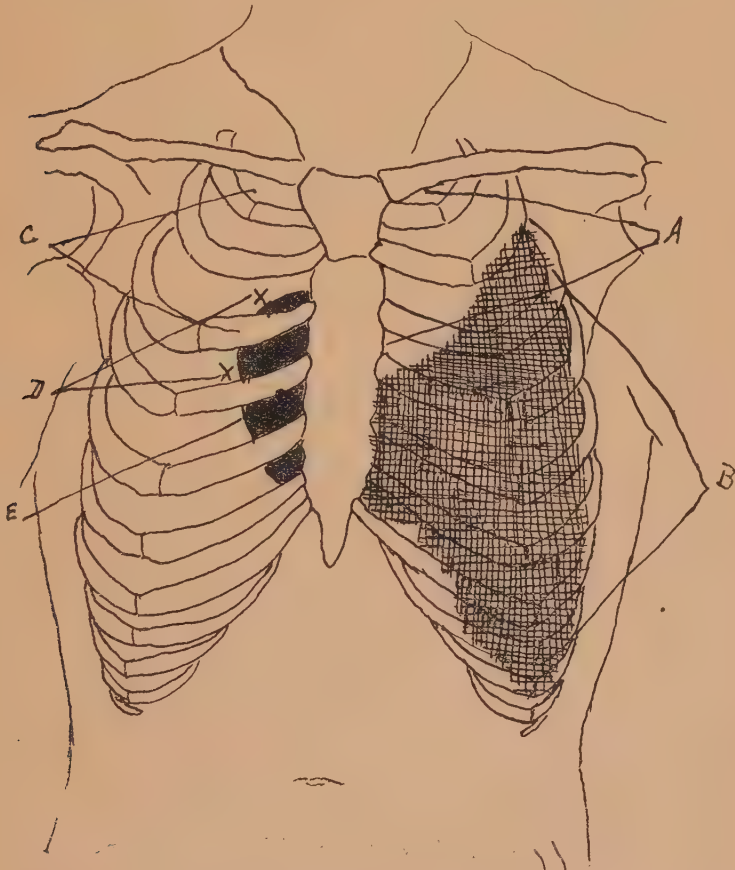
Physical signs in effusion into left pleura.

A.—Skodiac percussion resonance, exaggerated vocal resonance,
tubular quality of breathing. B.—Complete dullness, absence of vocal
fremitus, no respiratory sounds. C.—Purile respiration, prolonged ex-
piration, exaggerated percussion resonance. D.—More or less well
marked pulsation. F.—Displaced area of cardiac dullness. Upper
limit of shading indicates anterior line of Damoiseau's curve.

much depressed and the tympanitic note of Traube's semi-
lunar space may be obliterated. In very extensive effusions
the flatness may extend to the clavicle, and, transversely, be-

yond the opposite side of the sternum. According to Pitres, Damoiseau's curve is not observed in small effusions, and in moderate sized effusions the anterior and lateral portions of the upper border of flatness form a horizontal line, while the posterior portion falls abruptly.

Above the level of the fluid in cases of moderately extensive effusion the percussion note may have a peculiar quality of tympany—the so-called Skodiac resonance. This is obtained in the sub-clavicular region and gradually disappears into dullness in the axillary and mammary regions. In front this resonance may cover a triangular area with the sternal border as the base. Skodiac resonance may be obtained behind above the level of the fluid but not so clearly as in front. Even in cases of very extensive effusion a modified form of Skodiac resonance will be obtained in the infraclavicular region. This variety of resonance over the upper portion of the chest, with marked dullness or flatness over the lower portion, strongly indicates pleural effusion and becomes positive with alteration in the upper level of the flatness with change in the position of the patient.

With extensive effusions the tympanitic note in the first or second interspaces may change in pitch when the mouth is opened, due to transmission of vibrations from the primary bronchus (the tracheal tone of Williams). A cracked-pot sound may also be obtained in the same situation (Shattuck).

During the absorption of the fluid the dullness gradually descends, but remains, in some degree, over the lower portion of the chest even after the fluid has been entirely absorbed, and may persist for a long time in the event of adhesions or induration of the pleura occurring, or any lung condition which interferes with expansion.

In regard to the value of evidence obtained by percussion relative to the amount of effusion present in a given case, we may note that Garland has pointed out that stomach tympany may be obtained at the level of the sixth rib in the nipple line in the presence of large effusions of the left side, and that similar effusions on the right side may not appreciably lower the liver dullness. Powell maintains that the clinical evidence of the conversion of negative into positive intra-thoracic

pressure-though the extent of the effusion is (1) by the dull-ness extending above the third cartilage—the patient being in the sitting position, and (2) by the Skodiac resonance becoming tubular in quality, or extinguished. He thinks a negative pressure is necessary for the development of the full-toned Skodiac note, and that tubular, cavernous, or tracheo-bronchial resonance in the region of the sterno-clavicular angle is indicative of positive pressure, and of a greater degree of effusion.

By auscultation we may obtain valuable evidence of effusion through both the voice and the respiratory sounds. The voice sound, in large effusions, may be entirely absent below the level of the fluid, more particularly so over the front and sides of the chest. In large effusions we can almost always obtain a modified form of bronchophony just above the lower angle of the scapula, and in moderate effusions the voice sounds usually have the same character just above the level of the fluid.

Ægophony is sometimes heard over pleural effusion and was thought by Laennec to be diagnostic, but it may be obtained over cavities, or consolidated lung. It is thought by some to be of value in distinguishing localized effusion from consolidated lung tissue. It is a sign of little value in pleural effusion. In some cases whispering pectoriloquy, Baccelli's sign (pectoriloque aphonique), is clearly heard through the fluid by means of direct auscultation. It was thought by Baccelli to be diagnostic of serous, in contradistinction to purulent, effusions. In most cases of localized effusion, and in some cases of large effusion, high-pitched voice sounds can be heard over the area of effusion. While typical ægophony is not common in effusion the voice sounds often have a peculiar nasal twang or lisp.

The respiratory sounds are absent below the level of the fluid in typical cases of considerable effusion. In the early stages the respiratory murmur is more intense above than below the fluid, and as the fluid increases in amount the sounds below the fluid become tubular in quality and eventually are bronchial or disappear altogether. In front and at the sides of the chest the respiratory sounds are usually absent, while behind about the lower angle of the scapula there can usually

be heard a bronchial type of breathing, particularly on expira-
tion, as the cartilages of the larger bronchi keep them from
collapsing. These sounds are not so soft as the bronchial
breathing of pneumonia and do not seem to be so distant from
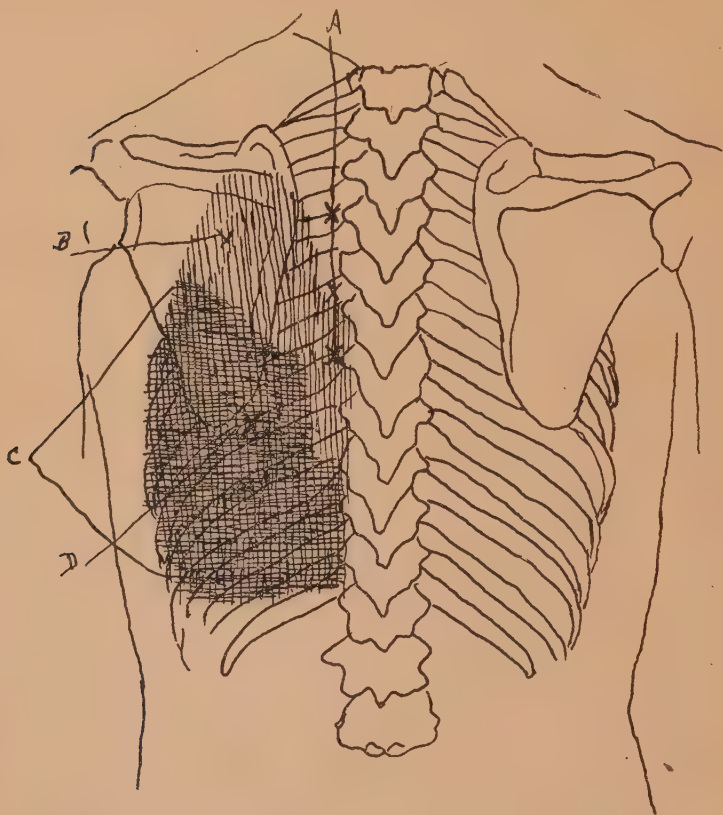
Physical signs (posterior regions) in effusion into left pleura.

A.—Diminished respiratory sounds. B.—Very faint or absent
voice sounds, absent breath sounds, dullness, no vocal fremitus. C.—
Dullness, no vocal fremitus, no voice or respiratory sounds. D.—Voice
sound is tubular or nasal in quality, sometimes approaching ægophony.
Upper limit of light shading indicates the posterior line of Damoiseau's
curve.

the ear. In localized effusions the respiratory sounds may be
high-pitched and clearly heard over the area of the effusion.
Bronchial rales, and fine, crackling rales from partial expan-
sion of collapsed lung may be heard on deep inspiration. In

some cases of considerable effusion fine crepitating rales may be heard on full inspiration due to congestion or œdema of compressed lung tissue. In moderate effusions friction sounds may be heard just above the level of the fluid, and where the pleura covering the tongue of lung which overlies the heart is involved there may be a pleuro-pericardial friction sound. The respiratory murmur in the opposite lung is increased in intensity—puerile type. The occurrence of congestion rales in the sound lung, together with the cough of congestion, blood-tinged expectoration, great dyspnœa and cyanosis indicates great pressure and a corresponding danger of syncope. When there is considerable displacement of the heart a systolic murmur may be heard at its base which will disappear with removal of a portion of the fluid. With small effusions the breathing may be tubular, metallic or amphoric in quality, and there may be loud, resonant rales suggestive of a cavity. As the fluid is removed the respiratory sounds gradually return to the normal, though they may be feeble and suppressed for a long time in the lower portion of the lung.

The diagnosis of sero-fibrinous pleurisy is usually not difficult in typical cases of moderate or large effusions. They are most likely to be confounded with pneumonia, but a careful consideration of both subjective and objective signs will prevent mistakes. In pleurisy the onset is apt to be insidious, the temperature is lower, the ratio of pulse to respiration is not distinctive, the cough is less troublesome, the sputum is not rusty or sticky, the surface of the body is not markedly hot or flushed, the affected side is enlarged, the heart is displaced, fremitus is absent, percussion dullness is more absolute even to flatness, voice and respiratory sounds are lessened or absent, bronchial breathing and bronchophony, if present, are feeble and not marked as in pneumonia, pneumonic crepitation is absent, the upper outline of dullness and its alteration with change of position are distinctive of pleurisy. Skodiac resonance is more pronounced in pleurisy.

In localized effusions the diagnosis may be difficult and only to be settled by the exploring needle. Pulmonary collapse, fibrosis, hydatids, and new growths in the pleura may simulate localized pleurisy. In all cases of doubt, exploration

should be made with the hypodermic needle. The use of the exploring needle has become almost a routine measure by many who would not admit doubt or lack of precision as to their methods of diagnosis. The information which it supplies as to the quality, not to say the nature, of the fluid may be useful from a therapeutic standpoint, and as it is a harmless measure when properly used, it should not be decried. In localized pleurisy the exploring needle is almost always necessary as the symptoms are so often indefinite we are seldom positive about the presence of fluid until we see it.

The signs to be considered in differentiating serous from purulent effusions will be considered hereafter, though the exploring needle is practically always relied on to determine this question.

TREATMENT.—In the treatment of sero-fibrinous pleurisy there is opportunity for the exercise of judgment with respect to the course to be pursued in the individual case. There is a somewhat widespread tendency to resort to aspiration in all cases of effusion which are susceptible of diagnosis. There is a subtle temptation in this method in that it enhances the patient's appreciation of the physician's skill as well as of his own moral and financial obligations in the matter, besides giving him the mental satisfaction of an ocular demonstration of the cause of his trouble as well as more or less physical relief. On the other hand the conservatism which advocates waiting a month or six weeks for the absorption of an effusion is not supported by our modern ideas of the ætiology of sero-fibrinous pleurisy. The views of H. I. Bowditch, the pioneer of the surgical treatment of serous effusions, which were considered somewhat radical in his day, are now regarded as correct.

Medicinally we may use hydragogue cathartics, diuretics and diaphoretics for the removal of effusions, but their action is uncertain and not much reliance is to be placed on them. Placing the patient on a dry diet and administering saline purgatives to deplete the serum of the blood, after the manner of Hay ($\frac{1}{2}$ to $1\frac{1}{2}$ ounces of Epsom salts in concentrated solution every morning or second morning before breakfast), may cause considerable effusions to disappear rapidly. Salty diet

may be allowed. Regular dosing with salt is recommended by some. Tonic medication is indicated and greatly favors the chances of absorption. Iodide of potassium and salicylate of sodium are recommended for the promotion of absorption and rather extravagant claims have been made for salicylate of soda in this respect. I have never been able to see that either of these remedies was of any particular value in promoting the absorption of pleural effusions.

There is as much difference of opinion as to the power of medicinal measures to promote the absorption of effusions as there is about the efficacy of antiphlogistic treatment in preventing effusion. Laennec, Guerin, and Peter believed in our ability to prevent effusions, while Rickard expressed his unbelief. Beaumetz, Dieulafoy, Montuard Martin, Faisans and others are non-committal. For the promotion of absorption Hayem, Köster, and Deri recommend salicylate of sodium. Huchard advocates the use of diuretics for the same purpose, while Rickard, Faisans, Talamon, Vincent, Harris and many others disclaim any faith in the power of drugs to promote absorption. Potain recommends sodium chloride in teaspoonful doses every few hours to prevent the reaccumulation of effusions.

The important question of which cases of effusion are suitable ones for aspiration relates mainly to free effusions. Localized effusions should always be aspirated when it is possible to do so as there is little prospect of the fluid absorbing until at least a portion of it is removed. An encapsulated effusion of only a few ounces may cause the persistence of pain, cough, dyspnœa and temperature elevation, and all of them are much relieved by the removal of the fluid. It is claimed that as these cases are usually tubercular the effusion acts as a splint to the lung and by lessening functional activity constitutes a conservative process which should not be interfered with. In many of these cases there is no history of tuberculosis, and such a condition cannot be demonstrated. The continued presence of a localized collection of fluid will result in changes in the pleura and lung which will permanently interfere with expansion. Even in cases of well advanced tuberculosis of the lungs the occurrence of an effusion may produce

symptoms which demand its removal, as occurred in the fol-
lowing case:

Young man 20 years old. Family history of consump-
tion. Sick ten months with tuberculosis of the left lung, the
upper lobe being consolidated down to the upper border of the
third rib. Temperature ranged from 99.5° to 101°. Became
worse rather suddenly. Temperature rose to 103° and pulse
to 120. Examination showed a lobular pneumonia of apex of
lower lobe. Pain in the side, cough and dyspnœa were dis-
tressing. These symptoms continued for more than a week,
the temperature not going below 102°. Examination showed
evidences of a local collection of fluid anteriorly below the
fourth rib on the left side. By aspiration eight ounces of fluid
was removed. The symptoms improved at once and in two
or three days the patient was quite comfortable.

In regard to free effusions we find many rules as to which
cases should be operated and when to interfere. The nature
of the fluid, the extent of the effusion, the age, personal and
family history of the patient are important in determining for
or against interference. In inflammatory cases with moderate
effusion, where friction sounds can be heard at the upper bor-
der of the fluid, and when the fluid contains many flakes of
lymph, it is likely that the fluid affords protection to the in-
flamed surfaces of the pleuræ and that the early removal may
favor the formation of adhesions. In these cases it is best to
depend on tonics and eliminatives for awhile and not to inter-
fere unless the fluid tends to increase rapidly, or in case it re-
mains stationary for two weeks.

According to Powell if there is good Skodiac resonance
down to the third rib the lung is in a condition of physiologi-
cal rest and interference is uncalled for; if pyrexia continues
at the end of the second week we should wait a week longer
before operating; if the patient's family history is unfavorable
we should interfere early unless the effusion is in the same
side with pre-existing lung disease; if the effusion extends
up to the second rib or higher, extinguishing Skodiac reso-
nance and causing decided increase in the measurement of the

side, especially if there is retching cough, frothy, viscid expectoration speckled with blood, and fine crepitations in the healthy lung, we should interfere at once without regard to the stage of effusion.

In a large percentage of cases of sero-fibrinous effusions of insidious onset the pressure symptoms as defined by Powell do not present, nor does absorption occur. While it is true that in most of these cases the presence of a considerable amount of fluid in the chest for three or four weeks does not necessarily compromise the lung to any great extent, it does the general condition of the patient. In view of the relief afforded the patient, as well as the avoidance of permanent injury to the lung by the early removal of a portion of the fluid, it is not clear that we are justified in waiting the appearance of pressure symptoms or of delaying for three weeks in the hope that absorption may take place. It is undoubtedly better to act within the shorter period stated by Bowditch, who says: "I cannot see any valid reason for continuing any active treatment more than one, two or three weeks without puncturing." This is all the more clear when we note the clinical history of these cases after partial aspiration, and how it emphasizes the force of Trousseau's observation that the "slowness in the absorption is, perhaps, as much dependent on the pressure exerted by the excess of fluid upon the serous membrane by which absorption has to be performed, as by the mere greatness of the quantity." The force of this observation is exemplified in the case of encapsulated effusions which, however small, are not absorbed, as a rule, until partially removed.

Free, sero-fibrinous effusions, especially those of insidious onset, should be aspirated as soon as there is sufficient accumulation of fluid to cause prostration, dyspnœa, much compensatory action of the opposite lung, and displacement of the heart; though, as before remarked, the latter symptom cannot always be regarded as directly proportionate to the extent of the effusion. Dieulafoy would aspirate an effusion of three or four pints, while Peter would not interfere with one of 1,000 grammes, but would aspirate one of 2,000 grammes, and Hardy operates if suffocation threatens. Peter would not operate until after the twentieth day, while Sée thinks effusion

is sure to return if not operated on before the twentieth or thirtieth day. Rickards thinks the effusion not likely to return if aspirated during decline of the temperature. Talamon thinks that a serous pleurisy which is not tapped is recovered from more completely than one which has been tapped.

No definite rule can be formulated regarding the time to interfere with an effusion. Each case presents its own indications. Subacute cases of insidious onset and with considerable effusion can usually be aspirated with advantage without special regard to the temperature or to the length of time the effusion has existed. The fever will generally decline rapidly after aspiration. In cases with more acute onset, high temperature, and exhibiting friction sounds denoting considerable plastic exudation, as well as considerable effusion, it is well to wait a week or two until the activity of the process subsides, before aspirating, as absorption of the balance of the effusion is not likely to occur during the active stage, and reaccumulation of the fluid is quite likely to take place, as occurred in the following case recently under observation:

Man aged thirty-three years. Laborer. Sick for a week with pain in the side, cough, dyspnœa and fever. Pulse 110; temperature 103°. Examination showed the left pleural cavity to be filled to the level of the fourth rib with fluid. Friction sounds could be heard from the third to the fourth ribs. Four days later there was no change in the amount of fluid and the temperature was the same. As the patient was attending the out-department of the clinic and was quite weak and suffering from dyspnœa, it was decided to aspirate the fluid although the activity of the process had not yet modified. Thirty-eight ounces of fluid were removed. Four days later the fluid had reaccumulated and the temperature was still high (102.8°). Ten days later the temperature had declined to 101° and the patient felt much better although the amount of fluid was fully as great as at the time of aspiration. Thirty-six ounces of fluid were now removed and recovery ensued without further trouble.

In the above class of cases the fever may be accepted as a rough clinical guide to the stage and intensity of the inflammatory process, and as such, may be taken as an indication for or against aspiration.

As to the dangers accompanying aspiration, they are, according to Potain, chiefly pulmonary congestion, syncope, embolism, and cardiac weakness. Samuel Gee points out the danger of death from serous expectoration immediately or shortly after operation and resulting from œdema of the lungs, also the danger of pneumothorax from puncture of the lung or from spontaneous rupture of the lung; of hæmorrhage from the pleural membranes, and of embolism of distant arteries, the most common result being hemiplegia. Pneumothorax may develop after operation, it rarely appears at the time. Subcutaneous emphysema may occur without pneumothorax. Free albuminous expectoration, associated with dyspnœa and likely to prove fatal, is described by French authors. It comes on after operation. These dangers are not imminent if aspiration is carefully performed. At the same time we must bear in mind the possibility of fatal syncope occurring in any case. The liability of transforming a serofibrinous effusion into a purulent one by aspiration was emphasized by Verneuil, Le Fort and others. Modern experience confirms the view of Beaumetz, Dieulafoy and others that this danger is insignificant with aseptically performed operations.

In encapsulated effusions it is evident that there can be no elective point for puncture. In free effusions we select the point at which we can most readily gain access to the fluid. This is usually in the fifth, sixth or seventh interspace just anterior to the axillary region, or about half way between the nipple line and the mid-axillary line. The interspaces are widest in this situation and the muscular tissues are thinnest. This region is most easy of access with the patient in the recumbent or semi-recumbent position. Another point often selected for puncture is posteriorly just below the angle of the scapula. There is more tissue in the way here, and the interspaces are narrower; there is likely to be a heavy deposition of lymph on this portion of the pleural membrane, and, accord-

ing to Gee, the lung is more apt to be adherent at this point, and the consequent danger of pneumothorax or of sloughing of the lung from perforation is greater.

Various forms of trocar and cannula, and reversible and rotary pumps, have been devised for the aspiration of fluids. The simplest means are the best. A trocar and cannula connected with a long tube in which is placed a glass coupling may be used. If nothing better is at hand an ordinary trocar and cannula may be used by fastening around the mouth of the cannula a piece of gold-beater's skin for a valve. When wet, this tissue will collapse and prevent the entrance of air. The best instrument is some aspirator like the Potain modification of the Dieulafoy instrument.

If the patient is weak or nervous a hypodermic injection of $1/6$ to $\frac{1}{4}$ grain of morphia may be given immediately preceding operation. If the heart is weak $1/30$ grain of strychnia may be given in addition. The patient should be in a reclining position with the shoulders elevated. In small, or encapsulated effusions, the sitting position may be best. Previous exploratory puncture will have told us how large a needle to use, though any needle may become blocked by plugs of lymph. The chest wall should be thoroughly cleansed. Local anæsthesia, and incision of the chest wall are unnecessary when using ordinary aspirator needles. If the tissues are indurated it often requires considerable force to get the needle into the pleural cavity, entrance to which is immediately appreciated through the lack of resistance. We should be careful not to injure the visceral pleura in moving the needle about, though some scratching of the pleura usually does no great harm. If a needle with a sliding point is used the danger of injury to the visceral pleura will be obviated. It is best not to employ any more suction force than is necessary to keep the fluid running.

There can be no rule as to the quantity of fluid to be removed. It is impossible to estimate the quantity of fluid in a given case, and we do not expect or desire to remove all of it. Aspiration should be stopped as soon as the patient exhibits hurried respiration, cough, or complains of pain. If the fluid becomes bloody, aspiration should be stopped. If lymph

blocks the needle or if deposits of lymph interfere with aspiration, the needle should be withdrawn and subsequent attempts made.

The withdrawal of a very small quantity is often followed by absorption of the fluid. If the fluid reaccumulates it should be aspirated as often as necessary. S. West advocates free incision and drainage in chronic cases of sero-fibrinous pleurisy which repeated tappings fail to cure.

The after treatment of these cases consists of tonic medication, pulmonary gymnastics, and, perhaps, change to a slightly higher altitude for a time. Restricted expansion of the lower portion of the lung may be present for a long time and some degree of loss of expansion may be permanent.

PURULENT PLEURISY.

Purulent pleurisy (empyema, suppurative pleurisy) is the result of an inflammatory process more intense but less sthenic than that which causes sero-fibrinous effusions. It is usually a secondary affection though it may be primary. Empyema may be acute or chronic, and may be general or local in extent.

ÆTIOLOGY.—Empyema arises from infection of the pleural membranes with some microörganism. Pneumococci, streptococci, and tubercle bacilli are the usual infective agents. Tuberculosis does not often cause empyema. When the latter condition occurs in tuberculosis it is likely to be due to pneumococcus or to streptococcus infection. The streptococcus is the common infecting agent in the purulent pleurisies of the adult resulting from the various septic infections of the pleura, and in empyema secondary to some of the infectious diseases. The pneumococcus is a common infecting agent in the purulent pleurisies of childhood. Meta-pneumonic empyema is almost always due to the pneumococcus, and these cases run the most favorable course. Pneumonia does not always precede the pleurisy. Primary pneumococcus pleurisy is a not uncommon condition. It must be remembered that pleural effusions after pneumonia are sometimes serous and may contain pneumococci. Also that serous effusions not secondary to pneumonia may be due to pneumococci.

The staphylococcus, the typhoid bacillus, the influenza bacillus, and other organisms may be the infecting agents in a few instances. In some cases observed by Thue during an epidemic of influenza, the pneumococcus was the organism most frequently found.

Empyema occurs as a sequence of acute sero-fibrinous pleurisy in persons whose general health is not good. It is difficult to explain the cause of this change in most instances. Aspiration is a rare cause of a serous effusion becoming purulent, and is constantly becoming more infrequent. Primary purulent pleurisy is not uncommon in children.

Secondary purulent pleurisy in common after pneumonia, scarlet fever, and typhoid fever; and more rare after measles, whooping-cough, diphtheria, erysipelas, etc. Local conditions such as wounds of the chest, fractures of the ribs, or perforations of the pleura by tuberculous processes in the lung frequently cause empyema. Pneumonia, bronchiectasis, pulmonary abscess, gangrene, cancer or tubercle; mediastinal diseases, pericarditis, diseases of the œsophagus, cervical abscess, cancer or abscess of the chest wall, tubercle of the bronchial glands, caries of the vertebræ, ribs, or sternum; peritonitis, subphrenic abscess, abscess of the liver, spleen or pancreas, and perforation from a gastric ulcer may act as causes of secondary purulent pleurisy.

MORBID ANATOMY.—Inflammations of the pleura which are of sufficient intensity from the start, or which develop sufficient intensity at any period, may be attended by the formation of pus. In this event the exudation of corpuscles is sufficiently free to give the exudate a purulent character. The effusion usually separates into clear, greenish-yellow serum in the upper portion, and thick, creamy pus in the lower part. It may be simply turbid and contain floculi of lymph. The pus from pneumococcus pleurisy is usually thick and creamy, is not clotted, and on standing does not readily separate into clot and serum, but thin, greenish serum appears on the surface after a time (Netter).

Pus from streptococcus pleurisy is moderate in quantity, yellow in color and not very thick. On standing it separates into two layers; the upper clear and abundant, the lower may

contain shreds of false membrane. Pus from an empyema usually has a sweet, heavy odor. In cases of wounds of the pleura the pus may be fetid. In gangrene of the lung or pleura the pus has a markedly offensive odor. The pus from an empyema may have a gelatinous consistence after withdrawal, or it may contain quantities of gas which makes it quite foamy in an aspirator bottle.

As the connective tissue of the pleural surfaces becomes involved in the process a granulating tissue is formed which may continue to generate pus. If the pus is drained its formation will generally cease, the granulation tissue becomes fibrous, and adhesions of the pleural surfaces occur. The pleural membranes may become greatly thickened, and form grayish-white layers from 1 to 2 mm. in thickness. The costal surface may show erosions and fistulous openings. The visceral pleura may show perforations. The new tissue in the pleural layers undergoes much contraction which is followed by various degrees of retraction of the chest walls. Calcareous plates may form in the adherent pleural layers and may be of considerable size. The lung may be markedly compressed. Displacement of the thoracic organs may be considerable. Intra-pleural pressure is usually higher in empyema than in serous effusions.

Loculated empyema (pleura abscess) usually occurs in the lateral or posterior, lower portion of the chest. It may occur between the base of the lung and the diaphragm, usually on one side, rarely on both. Interlobar empyema may occur and may be discharged through the lung into a bronchus. Apical empyema is very rare but may occur.

CLINICAL HISTORY.—The clinical history of purulent pleurisy is quite as variable as that of sero-fibrinous effusions. The onset may be abrupt with high fever, cough, pain and early dyspnœa. Quite frequently the onset is insidious and takes place during the course of, or following, some of the acute diseases. There may be no pain, little or no cough, and no dyspnœa until the pleural cavity becomes quite full. The character of the effusion and, to some extent, its clinical manifestations are largely determined by the virulence of the infecting organism and the resistance of the individual. Virulent

infections cause effusions rich in cell elements anl deficient in fibrin, and are likely to be purulent from the start. This is particularly true of streptococcus infections, though they may be primarily sero-fibrinous. Streptococcus pleurisies are most common in the adult after the infectious diseases, pneumonic inflammations, and may occur after pneumothorax from perforation in tuberculosis. The onset may be acute, with pain, high and irregular temperature, and repeated chills. Again the onset may be insidious with only moderate temperature. Absorption rarely occurs; they seldom discharge through the bronchi; reaccumulation after puncture is rapid and frequent; the course is toward chronicity, and relapses are common. The mortality rate is high (25 per cent.—Straus).

Pneumococcus pleurisies may be primary but are usually secondary to pneumonia, coming on with the onset, during the course of, or after the crisis of the lung inflammation. They may come on several months afterward (Straus). Usually they come on immediately after the crisis. The onset may closely resemble that of pneumonia with the exception of a more rapid pulse. In primary pneumococcus pleurisies there may be an imperfect crisis about the seventh day, the dyspnœa, however, persisting. The temperature in pneumococcus pleurisies usually ranges from 99° to 102°, and in some instances may be absent (Straus). The effusion may be primarily sero-fibrinous and subsequently become purulent. The prognosis is more favorable than in any other form of empyema. Absorption may rarely occur. Simple aspiration may be followed by recovery. If left untreated they are very likely to evacuate spontaneously.

The following histories are typical of meta-pneumonic empyema from pneumococcus infection:

Girl, aged seven years. Left apical pleuro-pneumonia. The pneumonia ran the usual course, being accompanied by marked pleuritic friction and pain. Crisis on the sixth day, the temperature going down to normal. On the evening of the seventh day the temperature rose to 102°. On the eighth, ninth and tenth days the temperature varied from 99.5° in the morning to 102° in the evening. Moderate sweating to-

ward morning. On the tenth day there were signs of fluid in the left side reaching as high as the third rib. Aspiration of over a pint of pus. On the twelfth day the fluid had reaccumulated. Siphon drainage instituted and maintained for four weeks. Complete recovery. Permanent loss of expansion less than one-fourth of an inch, as observed two years later.

Young woman, seventeen years old. Pneumonia terminating by lysis on the ninth and tenth days. Temperature normal in the morning, and 99.5° in the evening for three days. Temperature then rose to 101.5°, and varied from 99° in the morning, to 101.5° in the evening. Examination showed the left side to be full of fluid to the upper border of the third rib. Aspiration of thirty-four ounces of pus. Five days after, the fluid had reaccumulated. Siphon drainage instituted and maintained for four weeks. Complete recovery. Loss of expansion, observed two years afterward, one-fourth of an inch.

Tubercular empyema may or may not be preceded by evidences of pulmonary tuberculosis. It may follow tuberculosis of the bronchial glands. The effusion may be at first sero-fibrinous. It may be due to streptococcus or to pneumococcus infection. The onset may be acute or subacute. The course has a tendency toward chronicity. The effects of the effusion on the general condition of the patient, and the pressure effects are often inconsiderable. There may be little fever. The fluid will usually return after aspiration. In a large proportion of the cases of tubercular empyema the various operative measures undertaken for its relief fail of the desired result because of the inability of the lung to expand, and they are converted into cases of chronic empyema which furnish a varied and irregular clinical history.

Chronic empyema is essentially a surgical affection though the responsibility for its occurrence has been laid at the door of medical unskillfulness (Ewald). That this charge is not just is pointed out by Curtis who calls attention to the fact that in some cases the patient has followed his usual avocation for weeks while ill and before coming under observa-

tion, and that in tubercular cases it may not be possible to prevent the empyema from assuming a chronic form.

The history of chronic empyema may be simply that of loss of flesh and strength, some dyspnœa, slight irregular fever, and eventually signs of enlargement of the side with, possibly, pointing of the abscess in some of the intercostal spaces, as in the following case:

Boy, aged eight years, sick for four months with irregular fever, shortness of breath, loss of appetite and flesh. Left side bulging below the fourth rib in front. In the fifth interspace to the left of the nipple line there is a prominence over which the skin is tense and reddened. Fluctuation can be detected. Examination shows signs of fluid as high as the fourth rib.

Symptoms of septic infection are more or less marked in empyema especially in children. Sweating may or may not be pronounced. There may be little cough, and the fever may be very irregular. In chronic cases there will be various degrees of deformity from contraction of the chest walls.

If purulent effusions in the pleura are not interfered with they may affect various tissues within or without the chest. They may disappear by absorption in a very limited number of cases, the pus becoming inspissated, lime salts deposited, and the pleuræ greatly thickened. Perforation into a bronchus may occur, usually in loculated empyema, in which case pneumothorax generally results. Necrosis of the visceral pleura may occur and the pus may find its way into a bronchus without perforation of the latter, in which case pneumothorax is absent (Traube). Perforation of the chest wall may occur (*empyema necessitatus*), usually in the antero-lateral region of the chest (Cruveilhier); and generally in the fifth interspace (Marshall). The pus may break into the mediastinum, pericardium, œsophagus, stomach, or peritoneum. It may pass down along the spine and psoas muscle into the iliac fossa.

SYMPTOMS AND DIAGNOSIS.—The subjective symptoms of purulent pleurisy differ from those of the sero-fibrinous form chiefly in there being a more irregular, septic type of fever,

greater prostration, less cough, greater tendency to sweating, less dyspnœa unless the quantity of fluid is great, and little or no pain.

Leucocytosis is generally present in purulent pleurisies and may be marked.

The physical signs are those of general or loculated effusion and are similar to those of sero-fibrinous effusions. The main differences in empyema are the greater degree of enlargement of the affected side, particularly in children with free, purulent effusion; the greater tendency to obliteration and bulging of the intercostal spaces; the more frequent occurrence of œdema of the chest wall, and enlargement of the subcutaneous veins; a greater degree of displacement of the heart, liver, or diaphragm, due, according to Senator, to the greater weight of the fluid. The breath sounds may be loud and tubular over a considerable effusion, especially in children. According to Baccelli whispering pectoriloquy is not heard over a purulent effusion.

Pulsation synchronous with the heart's action is a phenomenon occasionally met with. Pulsating pleurisy occurs in two forms: The intrapleural form in which the whole side of the chest may pulsate without any bulging or pointing of the effusion. In most of these cases the pulsation is confined to the præcordial region or to the lower intercostal spaces (Gee). In the second form—the pulsating empyema necessitatus, there is an external, pulsating, bulging tumor in one or two places. The bulging is rarely larger than an orange. In either variety the heart is much displaced, and the heart sounds may be conducted to the bulging area. There is no thrill on palpation, and no expansion similar to that of aneurism. Pulsating effusions are usually chronic and practically always left sided, and may be associated with pneumothorax, in which case the pulsation is conveyed by the fluid alone. The effusion is almost always purulent, though pulsating, sero-fibrinous effusions have been observed. Loculated empyema may pulsate if situated near the heart. If so there is always bulging. Intrathoracic aneurism, and pulsating, cancerous tumor may simulate pulsating empyema.

In the differential diagnosis from sero-fibrinous effusions

the exploring needle will settle many doubts and prevent many
mistakes. The following characteristics of purulent effusions
may, however, be taken into consideration: They are most
common in children; most often due to pneumococcus or to
streptococcus infection; the onset may be of the pneumonic
type in primary pneumococcus infection or in meta-pneumonic
cases, while in the adult it may be insidious; in the later stages
the fever is of the hectic type, or, in some instances, the fever
may be absent; the clinical history shows emaciation, profuse
perspiration, febrile urine, rigors, anæmia, cachexia, and, in
adults, great prostration and delirium; leucocytosis is present;
fluctuation in the interspaces, œdema of the chest wall, point-
ing and pulsation may occur. In loculated empyema there
may be rupture of the sac. Absorption is rare but may occur
in pneumococcus infections in children.

In fetid empyema we may have typhoid, septic symptoms
indicative of infection of the whole body. Similar symptoms
may occur in cases which are not fetid. Coma and delirium
may be present. Enlargement of the spleen, diarrhœa, and
joint symptoms may occur. Death may take place in ten or
fifteen days no matter what treatment is employed.

Loculated diaphragmatic empyema may be associated
with hepatic or subphrenic abscess. It may be latent and is
difficult to diagnose especially when it contains gas. Sub-
phrenic abscess usually occurs on the right side. There is
usually pleurisy on the same side, generally empyema with or
without perforation of the diaphragm. The pus is fetid and
may burst into the lung even if there be no empyema. If em-
pyema be present it will be localized and the physical signs of
the two conditions will be practically identical. There is ful-
ness and tightness of the hypochondrium; the liver may be de-
pressed, but is not always so; besides, it may be depressed in
uncomplicated empyema. In subphrenic abscess there is dull-
ness and signs of effusion at the base whether effusion be pres-
ent or not, as the diaphragm may be pushed upward and the
lung compressed or collapsed. The heart apex may be dis-
placed without there being empyema, but usually is not.
Puncture or aspiration may show the presence of pus, but may
not indicate its location above or below the diaphragm as the

needle may pass through the diaphragm when the latter is much elevated. Even resection of a rib and exploration with the finger may be indecisive. When a subphrenic abscess contains gas the sounds may have a general amphoric character and there may even be no liver dullness.

In some cases of long continued abdominal pain the patient may develop a unilateral type of breathing resulting in an elevation of the diaphragm and of the liver dullness, laterally and behind, of as much as two inches, the anterior, lower border of liver dullness remaining unchanged. This area of dullness may resemble that of hepatic subphrenic, or localized pleural abscess.

Pyopericardium, suppurating hydatids of the liver, pleura, or lung; necrosis of the ribs, sternum, or vertebræ, Wunderlich's "peripleuritis" etc., are conditions to be eliminated in the diagnosis of localized empyema.

TREATMENT.—The treatment of purulent pleurisy is altogether surgical. The fact that it is possible in very rare instances for cases of pneumococcus infection to become absorbed has absolutely no bearing on the indications for surgical measures. We will not enter here into a discussion of the general surgical treatment of empyema, but will consider a few points of general interest.

In acute, general empyema the course to be pursued depends upon the nature of the infection, the age of the patient, and the character of the effusion. In children and young people general empyema occasionally recovers by simple aspiration, though this may have to be repeated several times. Upon one occasion I performed the ninth aspiration on a boy for post-scarlatinal empyema. He was aspirated twice subsequently. About a year afterwards I examined the boy's lungs and found it difficult to tell which side had been affected. It is generally admitted, however, that we are not warranted in trusting to simple aspiration for the cure of purulent effusion, and that drainage is necessary.

It makes but little difference what methods are pursued provided that drainage is sufficiently free, though there is much discussion over the procedures necessary to procure adequate drainage, particularly in relation to the necessity for rib

resection. Surgeons are generally in favor of resection in all cases of empyema. Godlee recommends resection "as a routine practice to which there are certain exceptions." The operation is neither difficult nor dangerous, and while there is no particular objection to it in any case, it is not necessary in every instance. In empyema in children general opinion seems to favor incision and drainage up to the end of the second year. After this period resection is generally recommended. Koplik recommends simple incision, at least for primary operation, in children under one year, especially when they are weak; in older and stronger children he thinks resection may be best. Holt recommends simple incision rather than resection. As the death rate in children from one to three years old is given as nearly fifty per cent. it is evident that the best results have not yet been attained. Bogart thinks that in children resection of the ninth rib in the posterior axillary line is ordinarily demanded, and should be preceded by a preliminary aspiration when the collection of fluid is large; primary irrigation, curettage, and multiple rib resection is contraindicated in children unless to aid in closing a persistent cavity or sinus.

Chloroform is not contraindicated in operating, though according to Winters full anæsthesia should not be induced.

In general cases of empyema secondary to infectious diseases, especially meta-pneumonic empyema, where by exploratory puncture we know that the effusion is comparatively thin and probably does not contain great masses of lymph, we can, by a simple siphon drainage, obtain more perfect results than by resection of the ribs. This method is but little more trouble than aspiration and does not in any way interfere with subsequent resection if the latter is deemed necessary. The cases whose clinical history is cited on pages 392 and 393 were siphoned in the following manner:

A trocar and cannula—the cannula constructed so as to just admit of the passage of 3/16 inch tubing—was passed through the sixth or seventh interspace in the anterior axillary line. The trocar was withdrawn and some 3/16 inch tubing passed into the chest and down almost to the bottom of the pleural cavity. The cannula was withdrawn over the tubing. The tube should project about eight inches from the

chest wall. Cotton is packed around the tube at its point of
exit, and a narrow strip of plaster fastens the latter to the
chest wall. Ordinary syringe tubing is convenient as that
portion of the tube which is within the chest becomes rigid and
does not flex on itself as pure rubber tubing is apt to do. On
the outer end of the tube there should be an ordinary flush-bot-
tle clip and a glass coupling. When the tube is in place about
three yards of tubing is attached to the coupling and run into
a jar of water at the bed side. With the thumb and finger the
air is then stripped out of several feet of the tube and the clip
is moved down and fastened below. After the pressure is
relieved the pus can be siphoned out rapidly or slowly by occa-
sionally stripping out a few feet of tubing and fastening the
clip. At the end of a week the tube can be withdrawn a couple
of inches, and by gradual withdrawal the cavity will become
small enough in three or four weeks to put in a short, open,
drainage tube, which can be removed and the opening allowed
to close when the discharge diminishes to about a dram in
twenty-four hours. A slight degree of chloroform anæsthesia
is desirable though not absolutely necessary in introducing the
tube. In a few days the patient can be placed in a wheel-chair
and taken out of doors, which is a great advantage in children
in hastening recovery.

General effusions of several weeks standing, especially if
there is tendency toward pointing, are better treated by free
incision with or without resection of a rib. Better drainage
is usually secured in all positions of the patient when the re-
section is made at the ninth rib just outside of the line of the
angle of the scapula (Gee), though many surgeons prefer
operating in the anterior axillary line. A chief danger after
operation is pneumonia of the opposite lung. Considerable
elevation of temperature after operation is usually due to im-
perfect drainage or to pneumonia. Caillé states that the tem-
perature remains near the normal after operation in very few
cases, but that its elevation always means something which
should be sought for and corrected.

Irrigation of the pleural cavity after operation for free
effusion should not, as a rule, be practised. Offensive effu-
sions generally become less so soon after free drainage is

established. There is danger of fatal syncope in some cases, and general opinion is against irrigation.

In localized empyema resection is almost always neces-sary. Irrigation is frequently indicated but should be used with caution. Irrigation by submersion in a warm bath is recommended by Zeman when the opening is large enough to allow the water to pass freely in and out. This method is endorsed by Adams. In tubercular empyema the propriety of establishing free drainage may be doubtful. In many of these cases it is difficult or impossible to close the cavity, and to produce expansion of the lung under these circumstances may hasten the destructive process in the lung. Repeated aspiration may be practiced, and sterilized or antiseptic solutions may be siphoned in and out of the cavity. In some cases of tubercular empyema operation may be indicated. In a recent case of tubercular empyema resection and drainage was instituted because aspiration relieved very slightly and only for a short time. The cavity was slow in closing but did so under irrigation. The activity of the tubercular process in the lung was increased subsequent to the operation.

In cases of double empyema it is safer to drain one side and aspirate the other if necessary, postponing the drainage of the opposite side until the patient becomes accustomed to the change in the respiratory conditions. In case there are adhesions it may be perfectly safe to drain both cavities at once.

Failure of an empyemic cavity to close is met with most often in tubercular cases. Absence of adhesions, extensive collapse of the lung, great rigidity of the chest wall, bronchial fistula, caries of the spine or ribs, tumors, and foreign bodies are the conditions which most commonly interfere with closure of the cavity. In tubercular cases where suppuration persists, it may be necessary for the patient to wear a tube for an indefinite period.

In some long-standing cases incision and drainage fail to result in closure of the cavity because the changes which have taken place in the pleuræ interfere with expansion of the lung, retraction of the chest wall etc., which are necessary to the closure of the cavity. These pleural changes are great thick-

ening ($\frac{1}{2}$ to 1 inch), and the development of calcareous plates which may be large or small and most often occur in tubercular cases. It may be impossible to remove them.

If a cavity fails to close in spite of free, single or double drainage, and particularly when the drainage is not perfect, the effects of chronic suppuration will appear: The patient becomes anæmic, exhibits hectic fever and develops amyloid visceral changes, enlarged liver, diarrhœa, emaciation and clubbed fingers; the urine is light in weight and may be albuminous. These conditions may almost entirely disappear if perfect drainage is established and the cavity closed. The possibility of attaining this desirable end by the performance of one of the more extensive operations, such as Estlander's operation should be considered.

In cases which exhibit, as a result of blood-poisoning, the so-called *"pulmonal cerebral abscess,"* the abscess should be opened if distinct localizing symptoms are present, as they are otherwise fatal. The connection of these abscesses with empyema is not clearly understood. According to Gowers they do not result from true tubercular cavities; the abscess is single in about half of the cases; it is usually situated in the posterior lobe of the hemispheres; the cerebellum is seldom affected and never singly.

CHAPTER XVII.

PNEUMOTHORAX.

By pneumothorax we mean the presence of air in the pleural cavity. The presence of air alone in the pleural sac is a rare condition, pneumothorax being practically always associated with serum (*hydro-pneumothorax*) or with pus (*pyo-pneumothorax*), and the term pneumothorax, as ordinarily employed, applies to either of these conditions.

Pneumothorax occurs more frequently in males than in females. It occurs chiefly in adults between the ages of twenty-five and thirty-five years—the period of greatest mortality from tuberculosis; but may be met with in very young children.

ÆTIOLOGY.—When air enters the pleural cavity the normal intrapleural tension in the pleural cavity (a negative force equal to from 6 to 8 millimetres of mercury—S. West) which practically equals, but opposes in direction, the normal elastic recoil of the lung (a positive force equal to from 6 to 8 millimetres of mercury—Donders) is relieved and atmospheric pressure is applied to both surfaces of the visceral pleura; the normal elastic recoil of the lung will then cause its collapse. This alteration affects the mediastinum, which is a movable partition, and its contents. The elasticity of the lung of the opposite side is also a factor in producing displacement of the organs within the chest. In explanation of the absence of pneumothorax in cases where it would appear to be a natural result, West assumes the presence of a cohesion of the pleuræ —equal to 12.5 millimetres of mercury—which counteracts the tendency toward retraction of the lung. If the lung on the affected side is adherent to the chest wall in part or in whole the elasticity of the lung is counteracted and the displacement of organs, dyspnœa, etc., will not be so marked.

Thus the effects of pneumothorax are more marked in a healthy chest than in one with adhesions between the lung and chest wall. If the pleura is entirely adherent and the elasticity of the lung completely negatived, the opposite lung may become considerably enlarged. Its elasticity, however, is not diminished, and it should therefore, according to West, be called "compensatory hypertrophy."

If the opening in the pleura by which the air gains entrance becomes closed or has a valvular action, the intra-thoracic pressure may become greater than that of the atmosphere, and the displacement of the organs within the chest may be increased. The rise in pressure may be due to rise in pressure from the valvular action of the opening during expiratory efforts, rise in temperature of the gas contained in the pleura, or to compression of the air by effusion into the pleural cavity.

Pneumothorax occurs from perforation of the costal, visceral, or diaphragmatic pleuræ; and from the spontaneous development of decomposition in pleural exudates.

Perforation of the costal pleuræ may arise from punctured or shot wounds of the chest wall, in which case it may or may not be associated with extensive emphysema of the chest wall; from exploratory puncture (Biggs, Osler); rarely from fracture of the ribs; and occasionally from abscess of the chest wall. Perforation of the diaphragmatic pleura from abscess originating in malignant disease of the stomach or colon, and especially from ulcer of the stomach, may cause pneumothorax. Perforation of the visceral pleura is the usual cause of pneumothorax. The most frequent factor in its production is tuberculosis of the lungs, especially acute pneumonic tuberculosis. According to various authorities ninety per cent. of the cases of pneumothorax are due to pulmonary tuberculosis. [Biach's collection of 918 cases shows 76 per cent. due to tuberculosis. Saussier's 131 cases shows phthisis in 81 cases, empyema in 29 cases, and gangrene in 7 cases.] The percentage of cases of tuberculosis which are complicated by pneumothorax is given by Powell and West as 5 per cent. and by Fowler as 6.5 per cent. Empyema and pulmonary gangrene are the next most frequent causes of pneumothorax by

perforation of the visceral pleuræ. Rarer causes are: abscess or hydatids of the lung or mediastinum, rupture of the pleura over a septic infarction, rupture of a hæmorrhagic infarction occurring in chronic heart diesase (Osler), rupture of air vessicles of a normal lung, (West thinks that an undetected lesion is responsible for these cases), strains from heavy lifting, or from unusual positions of the body, emphysema, bronchiectasis, disease of the œsophagus or bronchial glands, and malignant diseases of the lung.

Pneumothorax from decomposition caused by the gas bacillus (*B. aerogenes capsulatus*—Welch) is very rare, and is denied by some. The objections of Jaccoud, Fagge, Powell and others, which served to displace Laennec's simple or essential pneumothorax as being unscientific in the sense of being non-perforative, will have to be qualified so as to admit of a non-perforative form of pneumothorax in view of the evidence furnished by Lévy, May and Gebhart, Hamilton and others.

Pneumothorax may occur in persons who are apparently in perfect health, and as such cases generally recover, the lesions which induce the pneumothorax may not be discoverable.

MORBID ANATOMY.—In simple pneumothorax the pleural surfaces may present their usual glistening appearance. Usually there are the ordinary changes of acute or chronic inflammation. In perforative cases the opening in the membrane may be very difficult to find, but can usually be detected by forcing air into the main bronchus. There may be considerable lymph on the pleural surfaces and adhesions may or may not be present. Serum or pus may be present in the pleural cavity if sufficient time has elapsed, since the perforation took place, for their accumulation. On opening into the pleural cavity, or on inserting a trocar, there may be a jet of air strong enough to blow out a lighted match. On opening the chest the lung is found compressed, collapsed, or carnified; the mediastinum and contents are displaced towards the opposite side.

In acute pneumonic tuberculosis a sub-pleural necrotic area may perforate the pleura during a coughing spell, or, perforation may occur while the patient is at rest or asleep. In the more chronic varieties of tuberculosis the involvement of the

pleural membrane results in the formation of adhesions which are likely to prevent the development of pneumothorax, otherwise the occurrence of the latter condition would be much more frequent in pulmonary tuberculosis. In rare cases a cavity, or a sinus leading from a cavity, may rupture through the visceral pleura.

As a rule the affection is unilateral, and the left pleural cavity is involved more often than the right. (34:29, Fowler; 83:42, West; 7:1, Louis; 55:30, Walshe; 23:16, Powell; 2:1, Lebert, Regnaud, Wintrich, Weil. Laennec regarded the right side as most often affected.) The perforation, on either side occurs most frequently from the upper lobe than the lower lobe, the usual site being the mid-lateral aspect of the upper lobe. The middle lobe of the right lung is affected in a small proportion of instances. There is usually but one opening which is generally small, though it may be of considerable size. There may be several openings, and they may be confined to one lobe or may involve different lobes.

In pneumothorax from punctured wounds the gas will be of the same composition as the atmosphere; in perforations of the visceral pleura the gas will at first be the same as in the alveoli: $N=79.5$, $Co^2=4.38$, $O=16.0$. If the opening is closed the oxygen will disappear and the carbonic acid and nitrogen may increase. If the effusion present is fetid there may be sulphuretted hydrogen gas present.

In pneumothorax from punctured wounds or from the rupture of an emphysematous vesicle the opening may become closed and the gas absorbed in the course of four or five days. In tubercular pneumothorax there may be no effusion, and little or no inflammation. In the majority of cases, however, there will be a variable amount of pus of a yellow, greenish, or grayish color. It may be fetid. The effusion is usually serous at first, then sero-purulent and then purulent. It may be purulent from the first. Serous effusions may be clear, turbid, flaky, or blood-stained. The exudate may be gelatinous, or in some cases there may be exudation of lymph without any fluid effusion. There is no constant relation between the duration of the condition and the amount of effusion.

The effect of the collapse of the lung, resulting from the

pneumothorax, on the progress of tubercular disease in the
lung involved, has caused considerable discussion. In many
cases there is, temporarily at least, modification or arrest of
the tubercular process, though this is disputed by some
observers.

CLINICAL HISTORY.—The clinical history of pneumo-
thorax will vary with its type and ætiological relations. The
onset may be sudden with severe symptoms, or the pneumo-
thorax may be gradually and quietly acquired without special
disturbance. This difference in onset depends on the rapidity
with which the air gains entrance to the pleural cavity, and on
the functional activity of the lung involved. If the lung is
badly compromised by disease and is adherent to the parietal
pleura, both of the above factors will operate in modification of
the severity of the onset of the pneumothorax. In severe cases
the patient may experience a sensation as if something within
the chest had given way; there will be severe pain in the back
or in the upper part of the chest, and the patient may recog-
nize the displacement of the heart. There may be severe shock
with temporary heart failure, cold extremities, clammy per-
spiration, rapid pulse and respiration, and great mental dis-
tress. Dyspnœa is a marked feature, it is sudden and urgent,
and is mainly due, according to Murphy, to the interference
with the piston action of the diaphragm. The degree of dysp-
nœa will be in inverse ratio to the functional activity of the
lung involved. Death may soon follow the collapse of the lung
if the lung involved is the one chiefly depended on for respira-
tion. In a case recently seen in consultation pneumothorax
occurred during the course of a pneumonia, about the fourth
day. Its advent was marked by collapse, some pain and great
dyspnœa. The dyspnœa modified somewhat with the develop-
ment of compensatory action in the opposite lung, but was
troublesome until death which occurred twenty-four hours
after the pneumothorax developed. The patient was undoubt-
edly tubercular.

In latent cases there may be no history of the onset, the
pneumothorax developing without special disturbance, and be-
ing recognized only on physical examination. These cases are
likely to occur in tubercular pneumothorax, and, according to

West, may occur occasionally in healthy adults. A recurrent variety of pneumothorax has been described by Goodhardt, West, and Furney.

The following case illustrates a common development of pneumothorax in pulmonary tuberculosis:

Man aged 36 years. Primary apical tuberculosis of the left lung. Secondary involvement of lower, left lobe. Temperature, 100.5° to 102°. The patient who had been well enough to be up and about, woke up one morning feeling weak and depressed. His temperature was 99°, and pulse 120; respiration somewhat more rapid than usual, but no special dyspnœa. Examination showed a pneumothorax of the left side. Two days afterward the temperature had regained its usual elevation. A slight effusion followed the occurrence of the pneumothorax, subsequently both air and fluid were absorbed. The patient died about eight months afterward.

After an attack of pneumothorax the temperature may show a considerable drop if its range has been previously high. If it has not been high the drop will be less manifest. After the shock passes off the temperature will return to its previous range. Its subsequent range will depend largely on the nature of the changes in the pleura. In latent cases not followed by effusion, and where the air is rapidly absorbed, there may be no pyrexia. If pus forms in the pleural cavity there will be fever of a hectic type. According to Peters, and Williams, the temperature of the axilla on the affected side may be temporarily lower than that of the opposite side.

As pneumothorax occurs, in most instances, in cases of advanced lung disease, it may be regarded as an unfavorable manifestation, although it may retard the progress of the lung lesion in some cases. If the mechanical results of a valvular opening be not too marked, or if the pleural inflammation or the pressure from effusion tends to close the opening, recovery from the pneumothorax may ensue. In cases not followed by effusion the gas may be absorbed in a few days. Of thirty-nine cases cited by Powell from post mortem records, the longest duration of life was twelve months, and the shortest, ten

minutes. Pneumothorax occurring in a healthy person fre-
quently ends in recovery. Tubercular cases die within a few
weeks as a rule. West places the mortality of pneumothorax
at seventy per cent.

SYMPTOMS AND DIAGNOSIS.—In severe cases, besides the
subjective symptoms already detailed, the patient will be found
sitting, leaning forward with widely dilated alæ nasi, livid or
cyanosed face, and unable to articulate clearly. After the in-
tensity of the symptoms has passed off the intense dyspnœa
may be relieved even though the respiratory rate increase with
the accumulation of air within the pleural cavity. With a
respiratory rate of fifty-two per minute the patient may not be
conscious of difficulty in breathing. (Walshe).

On inspection in a case of general pneumothorax of con-
siderable extent the side will be found to be immovable and
enlarged. Its surface will be smooth from the absence of in-
tercostal depressions. The shoulder is raised. The heart im-
pulse may be displaced or may not be visible. If the pressure
is great there may be bulging of the chest and distention of
the superficial veins. In pneumothorax from perforation of
the visceral pleura by an empyema, particularly in chronic
empyema, and in cases due to perforation of the diaphragm-
atic pleura, if the opening is patent the side may be diminished
in size or retracted. In pneumothorax circumscribed by pleu-
ral adhesions, enlargement may be slight, some expansion will
be noticeable, and slight inspiratory retraction of some inter-
spaces may occur. The opposite side may develop compen-
satory enlargement, but the interspaces will show inspiratory
recession.

Palpation will determine the absence of the cardiac im-
pulse from its usual situation. Its actual situation is usually
evident, though it may not be. The displacement of the heart
occurs suddenly, may be recognized by the patient, and is an
important symptom whose relation to pneumothorax was first
pointed out by Gaide in 1828. Its sudden and early occurrence
shows that it is due to the removal of the elastic-traction force
of the collapsed lung and to the increased effect of the same
force in the opposite lung, rather than to pressure. The liver
and spleen may be depressed, the former may be as low as the

iliac crest. Vocal fremitus is absent over the affected side except in cases with pleural adhesions, when some fremitus may be felt.

Percussion over the air-containing cavity gives a tympanitic note which may be obtained beyond the mid-sternal line if the mediastinum and contents are much displaced. The note may be amphoric in quality; it may be hyper-resonant as in emphysema, of a flat quality similar to Skodiac resonance, or, in rare instances, dull.—"Muffled, toneless, almost dull." (Walshe). It is claimed that with extreme degrees of tension the note may be markedly dull, but this appears to me to be doubtful, though the pitch is often considerably raised. Auscultatory percussion may furnish a note of strongly metallic character. Displacement of the heart, liver, and diaphragm may be recognized by percussion. In left-sided pneumothorax the area of præcordial dullness is usually obscured, and the normal stomach tympany will interfere with our recognizing downward displacement of the diaphragm. It is claimed that a cracked-pot sound may be obtained if the opening is patent. if so, it is a very inconstant sound.

If fluid is present there will be dullness or flatness below, and tympanitic resonance above. The level of the dullness is more readily altered by a change in the position of the patient than it is in pleurisy. The upper level of a given amount of fluid may be much lower than in pleurisy, owing to the downward displacement of the diaphragm. Intercostal percussion may give a sense of fluctuation and a thrill. According to Walshe, the latter is most marked between the tympanitic and dull areas.

Auscultation of the respiratory sounds shows them to be suppressed on the affected side. Distant, tubular, or amphoric breathing may be present. In circumscribed pneumothorax with a free opening there may be loud amphoric breathing. Metallic rales may be present and metallic tinkling (Laennec) may be heard on deep inspiration, coughing, speaking, swallowing, or may occur from the movement of the heart. The voice sounds are amphoric or metallic in quality, and may have an echo-like character.

The so-called coin-sound or bell-sound (*bruit d'airain.—*

Trousseau) is a characteristic metallic echo which is one of
the most constant signs of pneumothorax. It is produced by
pressing a coin flat upon the chest, over the pneumothorax, and
tapping it with another coin while the ear or stethoscope is
applied to the opposite aspect of the cavity. If there is fluid

Physical signs in pneumothorax.

A.—Tympanitic percussion note, breathing suppressed, tubular or
amphoric, voice sounds metallic or amphoric; metallic tinkling. B.—
Dullness, absence of all sounds. C. Harsh breathing (upper portion),
increased percussion note and increase in pitch of voice and respiratory
sounds (lower portion). D.—Displaced area of cardiac dullness. E.—
Displaced spleen.

in the pleural cavity we may have, in addition to the above
signs, the succussion sound. (Hippocrates.) This splashing
sound has the peculiar metallic quality common to the other
signs of pneumothorax, and is caused by the splashing of

liquid effusion in the air cavity. It is obtained by shaking or jolting the patient while the observer's ear is applied to the chest wall. The patient may himself recognize the sound.

The diagnosis of pneumothorax is usually not difficult. The circumscribed variety is most likely to be confused with other conditions. If the percussion note is dull we may mistake pneumothorax for pleurisy. Diaphragmatic hernia may simulate pneumothorax, but usually has a history of injury. Very large cavities in the lung may present metallic tinkling and the amphoric qualities of voice and respiratory sounds peculiar to pneumothorax, but there is absence of dislocation of the organs, of the succussion splash unless there is an unusually large quantity of liquid in the lung cavity, and of the coin-sound, though the latter may be obtained in rare instances over a large cavity. Findlay has known emphysema to be mistaken for pneumothorax, and points to the bilateral nature of the former, also the absence of cardiac displacement, of the coin-sound, and to the less tympanitic percussion note as differential points.

Pyopneumothorax subphrenicus (Leyden)—a condition in which an abscess cavity beneath the diaphragm receives air through a fistulous communication with an air-containing viscus (usually from perforating ulcer of the stomach)—may simulate pneumothorax, but can usually be recognized by the history. If the similarity in the voice and respiratory sounds sometimes heard over the upper portion of the lung in cases of pleurisy, or over consolidated lung in pneumonia, to those of pneumothorax, confuses us, we may avoid mistakes by careful consideration of the general condition of the patient, the subjective symptoms of the disease, and the location of its physical signs. Subjectively, an asthmatic paroxysm, or hysterical dyspnœa, may resemble pneumothorax, but physical examination readily removes any doubt.

TREATMENT.—If the occurrence of pneumothorax is attended by severe pain, dyspnœa and mental distress, a hypodermic injection of morphia should be given. If severe shock results from the severity of the attack we may give hypodermic injections of ether. Strapping the chest has been recommended for the relief of pain and dyspnœa, and in some

instances may possibly be of service. Cupping and venesection had been advised for the relief of the pressure on the right heart in cases associated with lung disease. These measures, however, are seldom advisable or necessary.

Surgical measures are the only means of giving direct relief to the conditions resulting from pneumothorax. Various procedures have been suggested. Potain recommends the substitution of sterilized air for the air and fluid in the pleural cavity, and good results have been claimed from it. Evidently such a measure must have a limited application.

The surgical indications are much the same as in pleurisy. In traumatic pneumothorax from perforation of the parietal pleura, surgical or otherwise, there is no special tendency for the air to accumulate in the pleural cavity, and if sepsis is not induced the air may become absorbed in a short time. The use of the Tell-O'Dwyer apparatus for maintaining respiration and preventing collapse of the lung may be of great service in cases of acute traumatic pneumothorax.

In pneumothorax from a valvular perforation of the visceral pleura there may be rapid accumulation of air which produces great distress through collapse and compression of the lung and displacement of the intrathoracic organs. Aspiration of the air gives only temporary relief, and it may be advisable to introduce a small cannula and leave it in until the opening in the lung has closed. This must be conducted under strict antiseptic precautions. If these cases are tubercular there will usually be some serous or sero-purulent fluid present, but the air may be the chief difficulty.

A pure hydropneumothorax is very rare, but cases have been reported in association with hydatids of the chest. If aspiration is followed by increase in the pneumothorax and reaccumulation of the fluid, a free opening will probably be necessary.

In tubercular hydro or pyopneumothorax, if the fluid is slight in amount and does not tend to increase, and the patient is fairly comfortable, the case had better be let alone. If the amount of fluid is large and tends to increase, aspiration may be tried. If this is not satisfactory Godlee recommends passing two needles into the pleural cavity, one in front connected

with a bottle of boric acid solution at a temperature of 100°F., and one behind, which is connected with an aspirator, or which may be used as a syphon. The pleural cavity is drained and washed out with the solution which is also removed, as far as possible.

In all cases of pyopneumothorax, except some cases of tubercular origin, especially in cases showing septic effusion, incision and free drainage is indicated. According to Fraentzel, Senator, and others, the incision should be made close to the upper margin of the ninth rib just below the angle of the scapula. Irrigation may be advisable, but frequently is not best.

In pneumothorax resulting from the perforation of variously situated abscesses into the pleural cavity, or where gas results from decomposition, the treatment should be on the same lines as in cases of empyema.

HYDROTHORAX.

Hydrothorax (dropsy of the pleura) is due to non-inflammatory transudation of serous fluid into the pleural cavity. It occurs as a secondary process in many conditions. The fluid is clear, usually without floculi of lymph, and is markedly albuminous. The amount of albumen varies greatly, but according to analyses of Schmidt, Hoppe-Seyler, Lehman and others, is greater than in dropsical fluid from other serous cavities.

ÆTIOLOGY.—Cardiac failure from chronic valvular disease, and Bright's disease are the most common causes of hydrothorax. Dropsical conditions due to cardiac failure from any cause may involve the pleural cavities. Hæmic dropsies may be associated with hydrothorax. Hydrothorax may be associated with hepatic cirrhosis, mediastinal tumor, or with aneurism. Intrathoracic tumor may cause hydrothorax by pressure on the azygos veins.

MORBID ANATOMY.—Any degree of effusion may be present. In recent effusions the pleural membranes retain their smooth, glistening appearance. In long-standing effusions the visceral pleura is usually slightly thickened, and is of a

dull, grayish-white color due to exudation which changes into a fibrous film of unequal thickness similar to that found in the peritoneum in chronic ascites (Fowler), and probably due to slight inflammation of the serous membrane from prolonged contact with the fluid.

With long standing effusion and thickening of the pleura the lower lobe, or more, of the lung may be collapsed. The anterior margins of the lower lobe may be thinned out. In cases of general dropsy in persons with pleural adhesions of one side, the corresponding lung may be œdematous, and there may be hydrothorax of the other side.

CLINICAL HISTORY.—Hydrothorax, like other dropsical developments, may be exceedingly irregular. It may, or may not, be present in conditions which should, apparently, affect all of the serous cavities alike. In renal disease it is usually bilateral, and we would expect the same condition in connection with heart diseases in which it is often unilateral, and almost always of greater extent on one side than on the other. Cases of general œdema, with or without ascites, may, or may not, be associated with hydrothorax, and the latter may be present in cases which show little or no œdema. In nephritis following the infectious diseases, hydrothorax may come on suddenly in advance of symptoms of general œdema.

There will be a history of dyspnœa gradually or rapidly acquired. In chronic heart or kidney disease the gradual increase in the dyspnœa is likely to be attributed to the gradual failure of the heart, and if examination of the chest is neglected we may misinterpret the cause of the dyspnœa. Marked oppression, orthopnœa, or cyanosis may be present.

Again, a moderate degree of hydrothorax may add but little to the respiratory distress. This was illustrated in a case of cardio-renal disease in which several ounces of fluid had been aspirated from the left pleural cavity in order to relieve a severe dyspnœa. The dyspnœa was not relieved. The vascular tension being very high, a vaso-dilator was administered, and the dyspnœa was rapidly relieved and did not reappear to a severe extent although the fluid reaccumulated.

SYMPTOMS AND DIAGNOSIS.—In many instances the gradual development of hydrothorax allows of the patient

becoming accustomed to the interference with respiration. The physical inability incident to the chronic diseases with which hydrothorax is associated also modifies the evidence of its advent. The subjective symptoms are therefore less marked and more liable to be overlooked than they otherwise would be.

The physical signs are those of fluid in the pleural cavity, though because of the absence of any lymph deposit and of the lesser density of the fluid they are not so pronounced as in cases of sero-fibrinous effusions. The side of the chest is usually not enlarged. The heart is displaced but little, and not at all if there is equal bilateral effusion. Vocal fremitus is absent. The percussion note is dull but not so absolutely flat as in sero-fibrinous effusion. Whispering pectoriloquy may be heard. Tubular breathing can usually be heard, and the fine rales of œdema are usually heard in the lower portion of the lung on deep inspiration.

The clinical history of the case will establish the differential diagnosis.

TREATMENT.—The general treatment of cases in which hydrothorax occurs is not to be considered here. As far as the effusion is concerned it should be aspirated in any case of chronic heart or kidney lesion when there is sufficient fluid to interfere with the proper performance of respiration. In some cases of chronic heart disease hydrothorax may be the particular element which interferes with re-establishing the circulation. The same precautions should be observed as in aspirating sero-fibrinous effusions.

HÆMOTHORAX.

The term hæmothorax is applied to a collection of blood in the pleural cavity. The hæmorrhagic forms of sero-fibrinous effusions which may occur in connection with Bright's disease, cirrhosis of the liver, pneumonia, malignant types of specific fevers, and with cancer or tuberculosis of the lungs or pleuræ are not included under the designation of hæmothorax.

ÆTIOLOGY.—Injuries of the chest resulting in laceration of the pleura or lung, fractured ribs, injury of the intercostal

or internal mammary arteries, rupture of an intrathoracic vein may cause hæmothorax. Rupture of an intrathoracic aneurism may cause sudden and fatal hæmothorax. A pulmonary infarction may rupture into the pleural cavity. Scurvy, purpura, and conditions attended by hæmorrhagic diathesis may be associated with hæmothorax. Fatal hæmothorax has occurred from the rupture of an aneurism of the internal mammary artery after operation for pneumothorax (Fowler).

Clinical history.—In traumatic cases a great amount of blood may collect in the pleural cavity in a short time, and will be attended by marked symptoms of hæmorrhage. In other cases the bleeding may be slow, and no very active symptoms will develop. The rupture of an aneurism may precipitate an extensive and rapidly fatal hæmorrhage preceded by no premonitory symptoms such as may occur when an aneurism perforates the trachea or œsophagus, when there may be attacks of moderate hæmoptysis.

Syncope, pallor, coldness of the extremities, rapid breathing, labored dyspnœa, subnormal temperature, and feeble, rapid pulse mark the development of hæmothorax.

In traumatic cases the blood usually coagulates, the serum becomes absorbed, and the clot may finally absorb also. If septic infection occurs there is acute inflammation and empyema. Blood may remain for a long time uncoagulated in the pleural cavity.

Symptoms and diagnosis.—Aside from the subjective symptoms, which may not be very distinctive in cases of slow bleeding, the signs of effusion beginning at the base and extending upward, particularly when following the occurrence of an injury, are most important. Friction sounds are absent. If the blood has coagulated, the percussion note is hard and flat. The area of dullness may indicate a much larger effusion than is actually present. A previous diagnosis of intrathoracic aneurism will assist the diagnosis in rapidly developed cases not associated with injury.

Treatment.—When the source of the hæmorrhage is not known, the treatment should be expectant. It is not expedient to remove the fluid unless the severity of the dyspnœa

requires it, and then only sufficient fluid should be removed to give relief to the symptoms. In traumatic cases, if the bleeding has been comparatively slight and has apparently ceased, the treatment should be expectant. Where injuries involve blood vessels which can be located, they should be tied at once, and if necessary a portion of one or more ribs should be removed in order to get at the point of hæmorrhage.

CHYLOTHORAX.

Chylothorax (chylous pleurisy) is a collection of chylous fluid in the pleural cavity. The effusion may be found in the pleural cavity alone or it may occur in connection with chylous ascites. Chylothorax has been described as a form of pleurisy, but as the effusion is non-inflammatory in nature it should properly receive an individual description (Fowler).

Chylothorax is usually caused by obstruction or rupture of the thoracic duct. Obstruction of the duct at its entrance into the left subclavian vein by narrowing, thickening, thrombosis, tubercular disease, or new growths may cause chylothorax.

When there is obstruction, the thoracic duct may be distended in its entire course, and the lymphatics of the lungs, pericardium, and pleuræ may be distended. If there is perforation of the duct without obstruction the lymphatics may not show such distention. The pleural membranes may be white and opaque; the pleural lymphatics milky and arborescent. The lungs may present collapse, fibrosis, tubercular or other tissue changes.

The fluid may be present in one or in both pleuræ. It varies greatly in amount and contains fat in the form of an emulsion. Chemically it contains albumen, globulin, fat and inorganic salts; while microscopically there will be found small refractive granules and a few leucocytes. According to Sidney Martin the specific gravity of the fluid is about 1.022, and it consists of about ninety-one per cent. of water, and contains from eight to nine per cent. of solids, the fat amounting to about one or two per cent.

The clinical history is such as may pertain to the nature

of the primary lesion and is in no way distinctive as the nature of the effusion does not influence the character of the symptoms in any special way. Emaciation does not appear to be marked even in those cases where considerable fluid has been removed by repeated aspirations. Dyspnœa may be marked, and pain may be present when there is considerable effusion. The pulse is but slightly accelerated, and the temperature is usually near the normal.

The physical signs are those of pleural effusion and are not specially indicative of the nature of the fluid. The diagnosis will rest on exploratory puncture.

The prognosis, while influenced by the nature of the primary lesion, is generally unfavorable, though according to Cayley it is possible for anastomosis with the right lymphatic duct to be established in cases of obstruction slowly developed.

The treatment of chylothorax is symptomatic. Aspiration should be performed when there is sufficient fluid to cause the patient to suffer.

TUMORS OF THE PLEURA.

Tumors of the pleura may be primary or secondary. Primary new growths are rare in the pleura, but may occur in the form of fibroma, sarcoma, or the so-called endothelial cancer. Secondary growths are not infrequent as the result of extension from other tissues. They are usually cancerous or sarcomatous.

Secondary tumors of the pleura usually result from infiltration of the pleural membranes from malignant disease of the lungs, mediastinum, or of the chest walls. Secondary involvement of the pleura is frequent in cases of carcinoma of the breast, but may occur when the primary lesion is situated in any portion of the body.

There has been some discussion over the nature of primary cancerous tumors of the pleura, but as they cannot be distinguished histologically from other cancerous growths it is not worth while, from a clinical standpoint, to discuss the question. They may form flat, multiple nodules of a white color, connected by bands similar in structure. They may

show clusters of epitheloid cells, with round or spindle-shaped nuclei, lying in a fibrous stroma. Alveoli may present at the periphery of, or throughout, the growth, and may be lined with cylindrical or cuboidal epithelial cells. The growth may involve the bronchial glands, the mediastinal tissues, or may extend along the peribronchial tissues into the lung.

Any variety of sarcoma may occur in the pleura, but the round-celled variety is most common. Sarcoma may occur as large masses involving chiefly the parietal pleura, with a soft infiltration of the pleural membrane, or there may be a thick, fibrous layer involving the entire pleural membranes. Secondary involvement may occur in any or all of the adjacent tissues.

Tumors of the pleura may be essentially fibromatous, but most cases of this kind are probably examples of mixed sarcoma and fibroma.

The clinical history of tumors of the pleura may cover a period ranging from two to three months to one or more years. The course of primary tumors is generally rapid and limited by months rather than years. The onset is insidious as a rule. Pain is usually slight or absent, though it may be severe and persistent. Cough and dyspnœa on exertion are usually present. Malaise and loss of flesh may be early symptoms, but extensive emaciation is not reached. Moderate, irregular fever is present. The fever is usually not great unless some complicating condition is present. Albuminuria may be present. Œdema of the limbs, and gastric derangement and vomiting may be present. If the patient has been under treatment there may be a history of aspiration of albuminous, bloody serum, or of some other surgical procedure for the relief of the primary disease in the breast or other tissues.

Pleural effusion is frequently present and the physical signs of tumors of the pleura are very similar to those of effusion. The chest may be rounded, bulging, or retracted. The heart apex may or may not be displaced. Vocal fremitus may be diminished or absent, as may, also, the vocal and respiratory sounds.

Tumors of the pleura are chiefly confounded with pleurisy, from which they differ mainly by their slow onset and

absence of marked pain or fever. Paracentesis may show the absence of fluid, or if fluid is obtained, its removal is not followed by disappearance of the dullness, and by reposition of the heart if the latter is displaced. A hæmorrhagic effusion is not uncommon in connection with tumors of the pleura, while it is somewhat rare in tuberculous or pneumococcus pleurisy. Pressure symptoms are usually absent unless other structures than those of the pleura are involved. In some cases of chronic tubercular pleurisy the symptoms may much resemble the early history of tumors of the pleura, and the diagnosis may be difficult in this stage.

The treatment of tumors of the pleura is symptomatic. If fluid is present in the pleural cavity it should not be removed unless necessary, as its removal gives little or no relief, may be followed by distressing dyspnœa, and is usually followed by reaccumulation.

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